THE
LARYNGOSCOPE.

VOL. XLVIII DECEMBER, 1938 No. 12

AFFECTIONS OF THE CRICOPHARYNGEAL FOLD.*

MR. V. E. NEGUS, London.

An exact description of the anatomy of the mouth of the esophagus was written many years ago by Killian and also by Keith; yet now, 30 years later, no accurate description has gained a mention in the majority of anatomical text-books. It is desirable, therefore, to commence with some remarks about the normal cricopharyngeal fold and sphincter (see Fig. 1). Considerable importance is attached to this region by virtue of its specialized function. The clinical significance of the sphincter has been stressed repeatedly by Chevalier Jackson.

Anatomy and Physiology: The fold is produced by tonic contraction of the underlying cricopharyngeus muscle, which consists of the lowest fibres of the inferior constrictor muscle; according to Elze the fibres are part of the musculature of the esophagus (see Fig. 2). Anatomically, these fibres are in continuity with the upper and more oblique constituents of the muscle, and also with the circular fibres of the esophagus; but physiologically there is extreme dissimilarity in the function of the various components. While respiration proceeds, the pharyngeal musculature is relaxed and open, while the cricopharyngeus remains in tonic contraction. The object is to prevent air being inspired into the esophagus, where it would serve no useful purpose. Besides, any air sucked into the gullet would take away so much from the tidal volume designed for respiratory purposes in the lungs. During deglutition the superior, middle and inferior constrictors propel a

*Read by invitation before the Section on Oto-Laryngology, New York Academy of Medicine, May 18, 1938.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Aug. 25, 1938.

bolus of food by their peristaltic co-ordinated contraction, and as the wave passes downward the cricopharngeus relaxes to allow the food to enter the esophagus.

Disordered Action of the Sphincter: Even normal contraction of the muscle may become uncomfortable to a sensitive patient and a condition is sometimes met with which may be termed *painful contraction of the cricopharyngeus.* This may be relieved by mild sedatives or soothing electrical treatment; but, if persistent, stretching the sphincter, under direct vision, may be called for. The sphincter may exhibit *spasmodic contraction,* usually as a reflex response to some irritative cause,

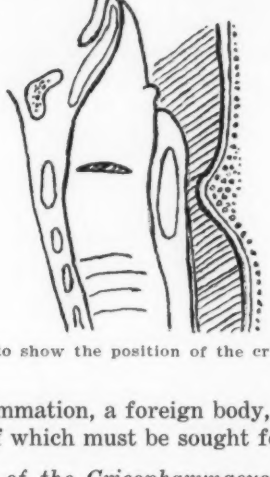

Fig. 1. Diagram to show the position of the cricopharyngeal fold.

such as local inflammation, a foreign body, or a stricture at a lower level, any of which must be sought for and treated.

Inco-ordination of the Cricopharyngeus may have serious results. If the muscle fibres fail to relax when a bolus of food is forced down — perhaps from lack of sensation in the posterior pharyngeal wall — considerable strain will be thrown on the walls of the pharynx. In front lies the larynx, at the sides are the alae of the thyroid cartilage and the fibres of the inferior constrictor, but posteriorly there is, during swallowing, a weak area between the circular and oblique muscle fibres (see Fig. 3). The weakness at this spot has been brought about by the evolutionary descent of the larynx in

man; it does not exist in animals for various reasons (see Fig. 4). The result of this deficiency may lead to gradually increasing herniation of the mucous membrane with, finally, the production of a pharyngeal diverticulum (see Fig. 5). In attempting to ameliorate or cure the condition the mechanism of production must be borne in mind; stretching the sphincter is required, in addition to other measures.

Impaction of Foreign Bodies: As reflex spasm of the sphincter is induced by irritation of the pharyngeal or eso-

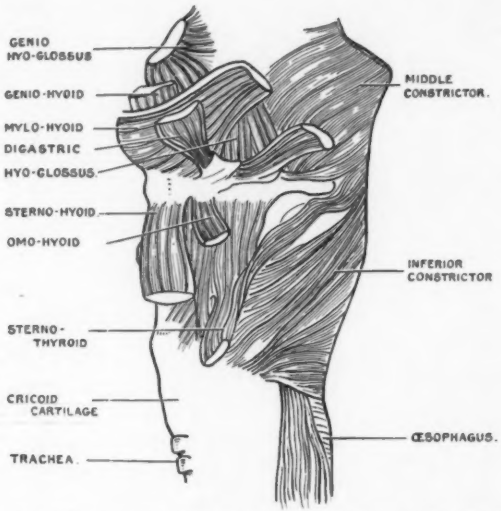

Fig. 2. Dissection of the pharyngeal constrictors and muscles of the hyoid bone.

phageal walls, this is obviously a favorite site for impaction of foreign bodies. For their safe removal relaxation of the sphincter is desirable, if tearing is to be avoided. It is my conviction that general anesthesia should always be employed when removing a pointed, impacted object from this region. Perforation may occur from contraction of the sphincter, which might force the esophageal walls over the point of a sharp object, such as a bone.

Webs of congenital origin and crescentic shape have been described; the symptom of dysphagia due to their presence may be relieved by dilatation.

Inflammatory Affections: As the fold forms a constriction on the food tract, it is necessarily the site of much friction.

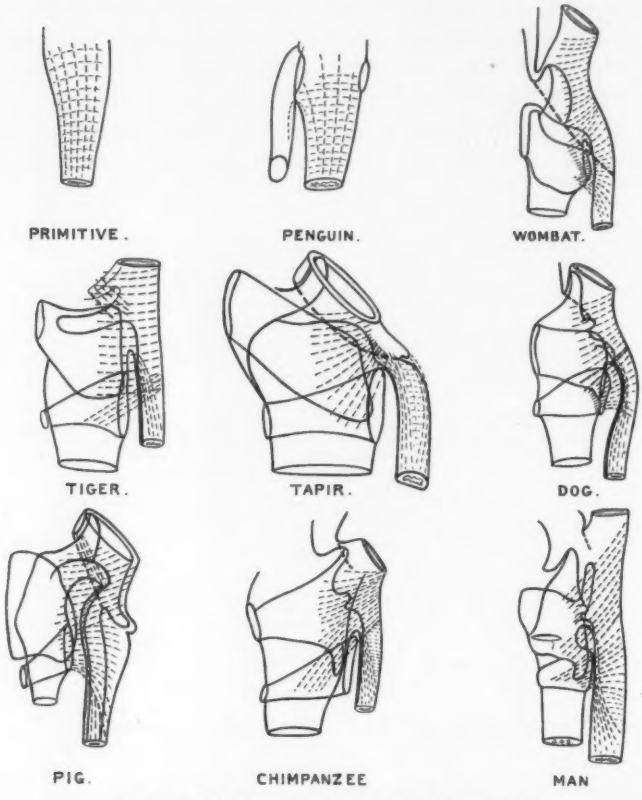

Fig. 3. Constrictor Muscles in Animals and Man.

The inferior constrictor is seen arising from the cricoid and thyroid cartilages of various mammals and man. For comparison, the simple arrangement of circular and longitudinal fibres, in a primitive type, are illustrated.

In a bird — illustrated by the penguin — the structure is also of simple type.

In the higher apes, and still more so in man, the larynx has descended to a lower level in the neck, and the muscle fibres are made to take an oblique course towards their insertion in the median raphe.

This change of direction is one reason for the presence of the weak area in the posterior wall, just above the cricopharyngeal sphincter.

It is, therefore, not surprising to find chronic inflammation of the mucosa. A satisfactory name for the condition is

"chronic hypopharyngitis" (see Fig. 6). This disease was described by Paterson and Brown-Kelly; it may be associated with superficial glossitis and secondary anemia, and is then known as the Plummer-Vinson syndrome. These inflammatory changes are usually limited to the upper or pharyngeal surface of the fold and appear as infiltration with superficial excoriation or ulceration, followed later by contraction and stenosis. In the later stages the mucosa is dry and glazed from atrophy and tears easily if stretched.

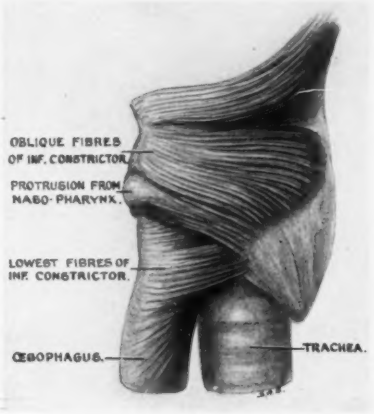

Fig. 4. Inferior Constrictor Muscle of a Pig (Sus Domesticus).

There is a tendency to herniation of the mucous membrane between some of the lower fibres of the inferior constrictor muscle.

Owing to the presence of an efficient arcus palatopharyngeus, all food is directed along the lateral food channel on either side of the larynx; there is, therefore, no tendency to enlargement of the potential pouch.

In man the posterior palatine pillar is short; there is no arcus palatopharyngeus, and nothing to prevent food being forced against the weak area between the oblique fibres of the inferior constrictor and the cricopharyngeal sphincter.

Middle-aged women are the usual subjects; they complain of slight difficulty in swallowing dry food, or a feeling as if something were stuck in the gullet at the level of the cricoid cartilage. Treatment includes wide dilatation of the fold, the taking of dry powders of bismuth, to form a protective and antiseptic covering over the abraded surface, and the administration of large doses of iron if there is anemia.

Carcinoma: Malignant growths at the mouth of the esophagus affect women much more often than men; the total num-

ber of cases is considerable, although the disease is not nearly so frequent as is carcinoma of the middle and lower end of the esophagus in men (see Fig. 7). When the patient is first

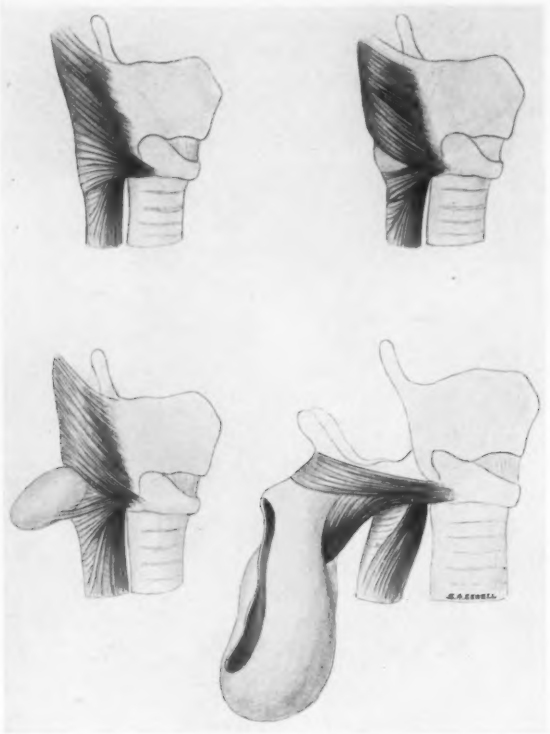

Fig. 5. Stages in the Formation of a Pharyngeal Diverticulum.

The top left-hand drawing shows the normal arrangement of the inferior constrictor muscle.

On the right is illustrated commencing herniation of the mucous membrane, between the oblique muscle fibres and the cricopharyngeal sphincter. (Prof. Parson's specimen in Museum, Royal College of Surgeons, England.)

Further stages are shown in the lower drawings. That on the left is from a specimen presented to the Royal College of Surgeons by Mr. Colledge, and the other is also in the Museum.

seen it is usual to find advanced disease, with an extensive growth spreading up behind the larynx, sometimes visible on indirect examination; occasionally there is extension upwards on the posterior pharyngeal wall.

The patient complains of discomfort or difficulty in swallowing, collection of saliva in the throat and possibly pain referred towards the ear. Further inquiry may elicit a record of slight difficulty in swallowing for years; sometimes the

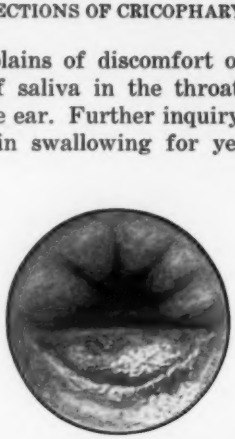

Fig. 6. Chronic Hypopharyngitis.

Chronic inflammation of the cricopharyngeal fold, on its pharyngeal surface in a woman, age 73 years. She had suffered from difficulty in swallowing for 23 years. Superficial ulceration is present and early malignant changes were suspected: the upper end of the esophagus was excised and the gullet was replaced by a skin tube, according to Trotter's method. The patient swallowed well, but later had stenosis, requiring a further plastic operation.

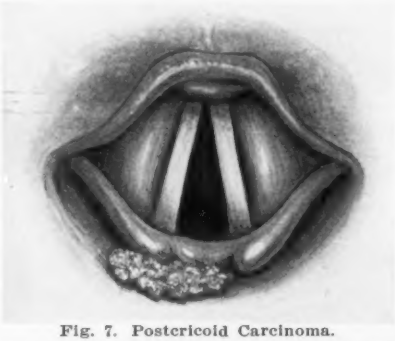

Fig. 7. Postcricoid Carcinoma.

A nodular growth is seen spreading upwards on the pharyngeal surface of the larynx. In the majority of such cases swelling of the arytenoepiglottic folds is produced, which, together with some impediment to movement of the vocal cords, causes hoarseness, together with dysphagia and pain.

patient observes that "she has always had a narrow swallow." It is possible, and in fact probable, that there has been previous hypopharyngitis in such cases. This is borne out by observations I have made on two or three patients in whom changes from simple inflammation to malignant invasion have

been watched in progress, repeated examination having been made over a period of 18 months to two years, or more (see Fig. 8). One patient had the upper end of the esophagus excised 25 years after the commencement of dysphagia caused by this hypopharyngitis.

The value of direct inspection in cases of dysphagia referred to the cricopharyngeal fold is obvious, as a malignant growth may thus be discovered in its early stages, while cure is still a possibility. There is a tendency with some practitioners to

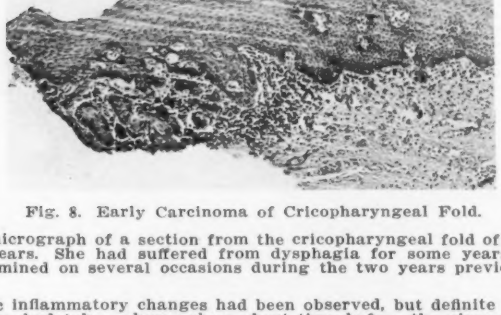

Fig. 8. Early Carcinoma of Cricopharyngeal Fold.

Photomicrograph of a section from the cricopharyngeal fold of a woman, age 37 years. She had suffered from dysphagia for some years and had been examined on several occasions during the two years previous to the biopsy.

Chronic inflammatory changes had been observed, but definite malignant infiltration had taken place only a short time before the piece was taken for examination. Healthy epithelium is seen to one side, and chronic inflammatory changes are present in one part of the submucosa. At the other end of the section there is commencing carcinomatous invasion of epithelial cells.

label the complaint of dysphagia as functional and due to spasm, on presumptive evidence which is quite insufficient.

If a growth is present at the mouth of the esophagus, and even if it extends well up on the back of the cricoid, it should be treated, in my opinion, by excision. If secondary glands are palpable, operation is usually undesirable, as cure is then unlikely; other contraindications are extensive involvement of the larynx by growth, cachexia and wasting and severe secondary anemia. The blood must be examined to discover whether hypopharyngitis has led to loss of red corpuscles and hemoglobin; the level may drop to so low a figure as 40 per cent.

Trotter's method of excising the esophagus, with replacement of the removed segment by a skin tube (see Fig. 9), is practicable in early cases and may be expected to give good results in a certain proportion. It is only feasible when the growth does not extend into the mediastinum, as it is essential to cut across the esophagus well below the lower limits of the lesion. Removal of the whole larynx does not appear to be necessary in well limited cases, but if the larynx is severely involved that organ must be sacrificed.

Both interstitial and distance radiation have been used for the treatment of postcricoid neoplasms, with success in some instances.

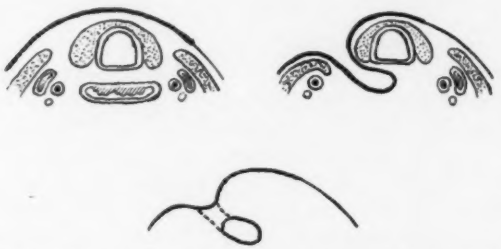

Fig. 9. Operation for Removal of a Segment of Esophagus.

The diagram represents Trotter's method of excision of the upper end of the esophagus and lower end of the pharynx; the cricopharyngeal fold is included in the segment removed.

The esophagus is shown lying behind the trachea, and the skin flap used for reconstruction of the gullet is indicated by a thick line.

In the right-hand figure the esophagus has been removed and the skin flap has been turned in to form a gutter.

The lower drawing shows how the gutter has been converted into a tube, to connect the lower end of the pharynx to the divided upper end of the esophagus.

The Cricopharyngeus After Laryngectomy: Esophageal speech has considerable connection with the cricopharyngeal sphincter and, therefore, a few remarks on the subject may be appropriate.

In complete removal of the larynx the fibres of the inferior constrictor must be carefully detached from their origin on the cricoid and thyroid cartilages; the cricopharyngeal sphincter is included in this detachment. When the pharynx is repaired, at the conclusion of the laryngectomy, the constrictor muscle is reunited so that it may regain its ability to contract and to propel food. It is to be presumed that the physiological

distinctions between the oblique and circular fibres remain, to a certain extent, and that a sphincter is thus reformed at the mouth of the esophagus. Apparently it has less control over the entrance of air than in the normal individual and so air-swallowing becomes easy. The faculty to draw air into the esophagus, and to store it there or in the stomach, is soon learned. When it is released the column of air passes from the esophagus into the pharynx. It appears probable that the constriction formed by the sphincter may even act as a reed and may thus be the origination of sound, for use in speech. The sphincter at the mouth of the air sac in certain fish, such as Gurnards (Trigla) is of equal simplicity, and is capable of throwing escaping air into vibration; for this reason certain varieties are known to fishermen as "crooners" and others as "grunters."

The Influence of the Sphincter on Esophagectasia: It has been noticeable, in my experience, to find an abnormally relaxed sphincter in the majority of cases of dilated esophagus, of the type generally known as cardiospasm or esophagectasia. The esophagoscope in such cases seems to slip into the gullet easily and one is led to search for the reason. In my opinion it is probable that undue relaxation of the fold plays a part in dilatation of the gullet. It may merely act as an aggravating factor, but may possibly be a primary cause in some, although not necessarily in all cases.

It is usual to consider the lower end of the gullet only as concerned in esophagectasia, the supposed causative factors being many. They include both spasm and lack of relaxation of the cardiac or diaphragmatic sphincter, due to excessive stimulation by the sympathetic nerve supply, or defective action of the vagus.

Periesophageal adhesions with kinking is another suggested reason. All of these views are based on obstruction as being the cause of dilatation. It is possible, however, for primary dilatation to occur, as a result of air-swallowing. If air is taken into the gullet and is carried through to the stomach, flatulence is produced; it is a common occurrence in infants. If the habit continues, a time will come when the stomach will hold no more air and some accumulation then takes place in the esophagus. If the amount is considerable the gullet will be distended; if the distension is repeated at frequent intervals,

or if the air is never gotten rid of completely, the esophageal walls will be stretched and the muscle fibres elongated (see Fig. 10). The process may continue for years before ill effects are noticed; the dilatation of the esophagus affects all parts and not merely the lower end where food accumulates. Eventually a time will come when the walls are so far apart that propulsion of food by peristalsis is no longer practicable. The patient has then to rely on the force of gravity for the

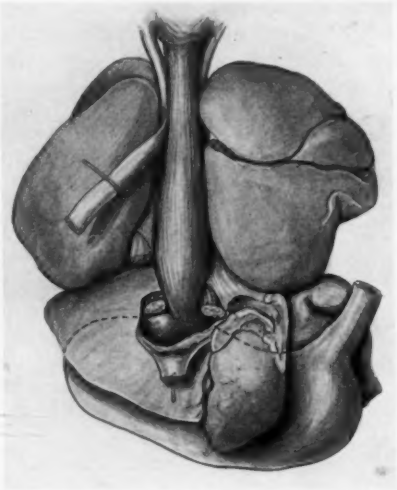

Fig. 10. Esophagectasia in an Infant, Age 6 Weeks.

The child had difficulty in swallowing, and died from lack of nourishment.

Postmortem examination showed an absence of hypertrophy, or atrophy of the esophageal walls, or musculature; no periesophageal adhesions in the liver tunnel; and no degeneration of Auerbach's plexus.

passage of food into the stomach. Separation of the walls of the thoracic esophagus is aided by the reduced pressure in the chest, while the abdominal portion is kept closed by mutual apposition of the viscera, thus causing obstruction to the entrance of food.

It would, of course, be possible for an individual with a normally contracting cricopharyngeus to acquire this air-swallowing habit, but it seems probable that the practice sometimes originates in infancy and that it may be associated

with undue relaxation of the sphincter, which then fails to carry out its function of preventing entrance of air into the esophagus.

Conclusions: These are some of the clinical lessons we may learn from an appreciation of the anatomy and physiology of the cricopharyngeal fold. They may be of help in suggesting remedial measures for the cure or relief of our patients.

I wish to express my gratitude to Dr. Chevalier Jackson, whose stimulating teaching first made me realize the importance of this subject. I am also grateful to Sir St. Clair Thomson, who has most kindly read through this paper and has offered many criticisms and corrections.

N.B.: The literature on the subject is so extensive that I have found it impracticable to include references. I apologize for this omission and, at the same time, I wish to thank the many authors whose writings have been consulted.

THE AMERICAN BOARD OF OTOLARYNGOLOGY.

An examination was held in Washington, Oct. 7 and 8, 1938, prior to the meeting of the American Academy of Ophthalmology and Otolaryngology. One hundred and twenty-nine candidates were examined; of this number, 97 were certificated.

During 1939 examinations will be held in St. Louis, May 12 and 13, prior to the meeting of the A.M.A., and in Chicago, Oct. 6 and 7, preceding the meeting of the American Academy of Ophthalmology and Otolaryngology.

Prospective applicants for certificates should secure application blanks from the Secretary, Dr. W. P. Wherry, 1500 Medical Arts building, Omaha.

DIPHTHERITIC STENOSIS OF LARYNX; COMPLETE LOSS OF VOICE, CURE BY IN-LYING BOUGINAGE CONSISTING OF SERIES OF METAL BEADS.*

DR. E. LEE MYERS, St. Louis.

Diphtheria in the last decade has been characterized by fewer complications. This is doubtless due to the profession's alertness, both as to the prophylaxis and the treatment. When diphtheria involves the larynx the situation is made worse because of the toxemia, as well as respiratory embarrassment. O'Dwyer's epochal discovery of intubation has saved many lives, yet in the occasional case the intubation tube may have been a factor in the production of an irritation from which a stenosis is made by opposing ulcerated edges. Complete stenosis of the larynx, while rare, is sufficiently met with to cause the laryngologist considerable concern. Jackson† cites 21 cases of chronic laryngeal stenosis which were waiting for him in Philadelphia, most of these cases being caused by faulty tracheotomy. No effort will be made to call attention to the work of Schmiegelow or any work that entails a cutting operation into the larynx from the outside. The following procedure, all of which was done endolaryngeal, proved to be entirely satisfactory in one case.

Case Report: Female child, age 3 years, became ill with a sore throat and fever four days before being taken to Isolation Hospital, Dec. 15, 1935. At the hospital she was given 2,000 units of diphtheria antitoxin by vein and 18,000 units intramuscularly. There was a slight respiratory difficulty on admission, with a history of having had a coughing spell the previous night, so severe as to almost cause choking. She presented a retraction of the substernal region, supraclavicular and the intercostal spaces, accompanied by a definite stridor both on inspiration and expiration. The following morning she was intubated; the tube being coughed up within two days. Difficulty in breathing made it necessary to replace it in eight hours. Tube remained in place eight hours, when

*Preliminary Report, St. Louis Medical Society Case Demonstration, Feb. 8, 1938, St. Louis University Unit, City Hospital.

†Page 193, "The Larynx and Its Diseases," Chevalier Jackson.

it was again coughed up and had to be reinserted within four hours. The tube was coughed up for the third time after 12 hours and was reinserted again within two hours, with immediate expulsion of same. A further attempt also resulted in immediate expulsion, and it was necessary to tracheotomize the child within a period of three hours. She proceeded to recover from the diphtheria, but it was impossible to remove the tracheotomy tube. A period of practically seven months was spent trying to remove the tube, but breathing was impossible without it.

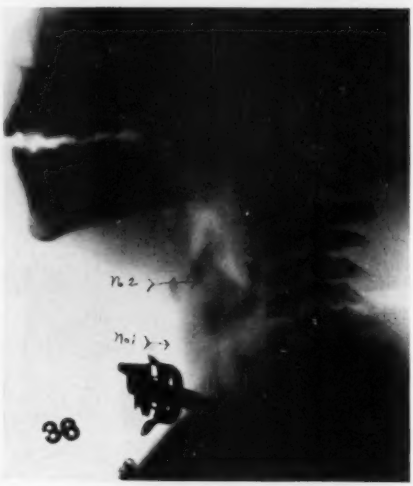

Fig. 1. Just above tracheotomy tube can be seen an area which stops at arrow No. 1. The space between arrows Nos. 2 and 1 is approximately the area of the stricture.

April 22, 1936, child referred to me by Dr. J. A. Rossen. Patient at that time was perfectly healthy except for the fact that she could not breathe without the tracheotomy tube. She was unable to utter a word, and no air was evident coming from either the nose or mouth on expiration. An X-ray showed a definite stenosis of the larynx.

Operation: Under avertin anesthesia at the Jewish Hospital, a direct examination with the Haslinger directoscope showed the larynx to be filled with granulation tissue. An

effort was made to enter the larynx with a probe. The tissues felt hard and unyielding; a long laryngeal knife was inserted

Fig. 2. Larynx is exposed, cords sealed together. This area when probed felt very dense.

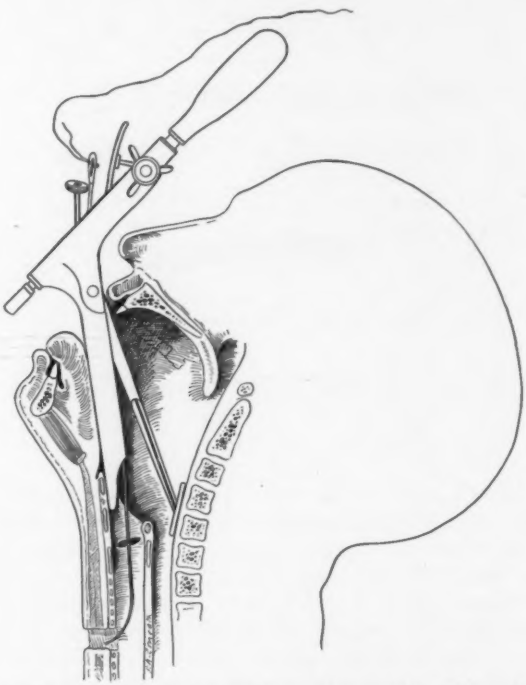

Fig. 3. Under avertin, uterine probe was inserted into the trachea after cutting laryngeal stricture. String was brought through tracheal wound and left in for subsequent work with braids and beads.

close to the anterior border of the larynx, the knife was gently pushed downwards for a distance of possibly three-fourths

inch, when no further resistance was felt. A long probe (uterine) successfully entered the trachea and was picked up by Dr. Eimer in the tracheotomy wound. The thread which was tied to the eye of the probe was pulled down the trachea and out of the tracheal opening; the upper (laryngeal) end was carried through the nose and tied to the tracheal end after hiding as much as one could by tucking it behind the ear.

The child was now transferred to the City Hospital for treatment. Our next problem was how to keep the strictured larynx open. At weekly intervals strands of braided silk were

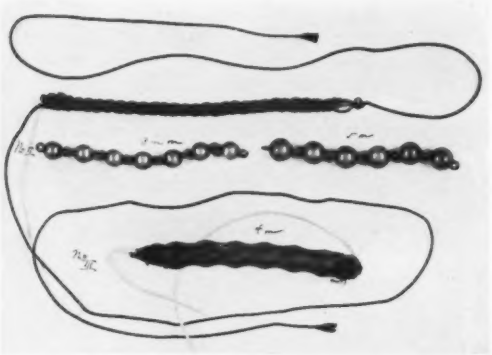

Fig. 4. 1. In-lying bougie made of a braid consisting of 12 strands. 2. Silver connected beads, 3 mm., 5 mm. 3. Four mm. beads covered with catheter tubing.

used, adding a strand each week and braiding the strands. At one time the braid contained 14 strands. Upon removal of the braid no air was noticed coming through the larynx. An in-lying bougie made of connected silver beads of three different diameters, 3, 4 and 5 mm., was then used. Weekly, under avertin, these beads were pulled down into place and allowed to remain in the larynx between treatments. Up to February, 1937, a continual change of beads was made, with no improvement. The larynx, while apparently open, did not seem to function. At this point the 4 mm. beads were covered with catheter tubing and the bougie was allowed to remain in the larynx for a month.

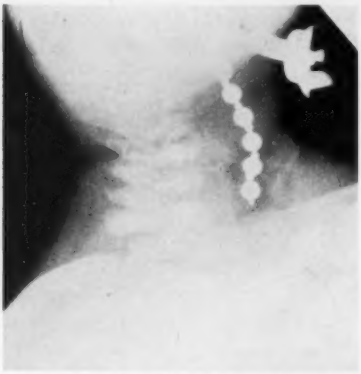

Fig. 5. Showing 4 mm. beads in situ.

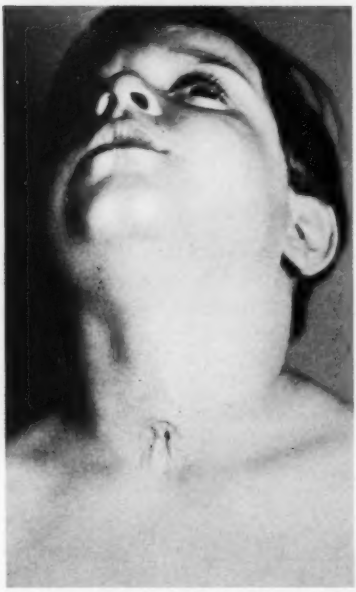

Fig. 6. Showing child with tracheotomy tube removed. Practically cured of her condition. Wound still leaks some but has not been repaired. This will be done at a future sitting.

March 2, 1937: The 5 mm. beads were covered with catheter tubing and left in the larynx for two weeks. April 20, larynx was bougied with Nos. 16, 18 and 20 triangular bougies; beads

were not replaced. May 11: Larynx appeared fairly normal; Nos. 16, 18 and 20 bougies were passed and beads were not replaced.

During all this time the child had not been able to utter a sound, even the faintest whisper was impossible. One morning while making his rounds the resident laryngologist was happily surprised to hear this child scream loud enough to be heard across the ward.

June 4, 1937: For the past two or three weeks child had been walking around the ward with a rubber cork in the tracheotomy tube, talking in a rough voice but breathing all right through the larynx; at night she would not trust herself to breathe through the tracheotomy tube with the cork in place. One day she removed her own tracheotomy tube and from then on the tube has not been needed. At the present time the child has a very good voice and on inspection larynx is gradually assuming normality.

A year of peroral laryngeal treatment, using a method patterned after the treatment for esophageal stricture, has obviated the necessity of splitting the larynx. The connected silver bead in-lying bougie which was used in this particular case seems to justify our efforts.

I wish particularly to thank Dr. A. P. Rowlette, of the St. Louis City Hospital, for his co-operation in providing the bougies, also Dr. Borg, the resident laryngologist, now of Chicago, and the nursing staff for their painstaking care.

207-8 Wall Building.

RADIUM THERAPY FOR RECURRENT EPISTAXIS IN HEREDITARY HEMORRHAGIC TELANGIECTASIA.

DR. J. ALLAN WEISS, Chicago.

The disease entity characterized by bleeding, usually from the nasal mucosa, by the presence of numerous telangiectases and by a familial and hereditary predisposition has been designated as in the title, or by various synonyms, such as multiple hemorrhagic telangiectasia, familial epistaxis, Rendu-Osler-Weber disease, heredofamilial angiomatosis and other combined terms. The literature presents reports regarding some 90-odd families, comprising about 550 individuals.[1, 2]

Of the many therapeutic measures employed in this condition, only a few have had any effect in controlling the repeated, severe hemorrhages. In cases of epistaxis, satisfactory results have been reported after the use of radium in amounts ranging from 50 to 1,200 mgm. hours on each side of the nose.[3-7] The following case illustrates an indifferent outcome after a moderately large amount of radiation therapy:

History of Case: P. S., male, age 68 years, was seen for the first time in June, 1935, in the Mandel Clinic of the Michael Reese Hospital. He had been subject to recurrent nose-bleeds since early in adult life. In the preceding year, bleeding had occurred at short intervals. Frequently the hemorrhages were profuse and difficult to control. One of his five children was afflicted similarly. No accurate information could be elicited regarding the remainder of his family residing in Poland.

On examination, multiple, flat, red areas ranging up to 2 mm. in diameter were found on the skin of the face, thorax and trunk, and on the mucous membrane of the mouth and nose. The lesions were most numerous on the septum and turbinates; they were present also on the tongue, soft palate, conjunctivae and buccal mucosa. A few larger varices were noted on the face, especially on the eyelids. Bleeding from the nose followed slight trauma, such as contact, forcible blowing or sneezing.

There were no important changes in the ears, larynx or pharynx.

The laboratory reported a hemoglobin of 70 per cent (Tallquist), a red blood cell count of 3,900,000 and a white blood cell count of 7,200, with a normal differential picture. The blood platelet count, the bleeding time and the coagulation time were normal. Urinalysis and the blood Wassermann were negative. The blood pressure was 140 systolic and 70 diastolic. Examination in other departments indicated a generalized arteriosclerosis with chronic myocarditis and angina pectoris, and also a chronic iridocyclitis.

A diagnosis of hereditary telangiectasia was made on the basis of the presence of typical lesions, the history of repeated epistaxis, the familial

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Sept. 23, 1938.

occurrence and the absence of the abnormal blood findings of a blood dyscrasia.

During the period of observation up to January, 1937, epistaxis occurred at intervals of three days to two weeks. When the bleeding was profuse, local application of various vasoconstrictor solutions was entirely ineffectual. Firm packing of the nose for a period of 24 to 48 hours was usually necessary. Premature removal of the pack often necessitated reinsertion. On some occasions cauterization of the active bleeding point with 50 per cent trichloracetic acid sufficed to arrest the hemorrhage; however, this did not prevent recurrences. Furthermore, it was obviously impossible to destroy all of the lesions present in the nasal mucosa by chemical cauterization. For this reason, radiation therapy was decided upon.

Teleradiation was administered with a 4 gm. radium pack at 10 cm. distance, with a 8 x 10 cm. portal and 1 mm. platinum plus 1 mm. copper filtration. This was supplemented by 25 mgm. intranasal applicators with a filter of 1 mm. platinum. The following schedule was carried out:

Jan. 1, 1937: Intranasal applicators; 100 mgm. hours in each side of the nose.

Feb. 4 to Feb. 18: Radium pack; 8,000 mgm. hours on the right side of the nose; 6,000 mgm. hours on the left side.

May 22 to May 30: Radium pack; 6,000 mgm. hours to each side.

Jan. 1 to Jan. 19, 1938: Radium pack; 6,000 mgm. hours to each side.

Jan. 31 to Feb. 2. Radium pack; 3,000 mgm. hours to each side.

Feb. 16: Intranasal applicators; 50 mgm. hours to each side.

The low-grade secondary anemia was treated with iron and ammonium citrate and a high-vitamin, antianemic diet. The degree of anemia was variable, depending upon the amount of recent blood loss. In June, 1938, the hemoglobin was 60 per cent, and the red blood count 3,650,000. A transfusion of 350 cc. citrated blood was given, with a resultant increase to a hemoglobin percentage of 70, and a red blood count of 4,250,000.

COMMENT.

The treatment given totaled 300 mgm. hours of radium intranasally, and 40,000 mgm. hours by teleradiation. This amount of radiation is comparable to the highest dosage reported.[4] During the summer months the frequency and severity of the epistaxis were noticeably decreased, but in the fall and winter the incidence approached that noted before irradiation. This variation is consistent with the fact that bleeding in this condition does not occur as often during the warm weather when the individual is less subject to nasal or upper respiratory inflammation.

It was to be expected that the fibrosing effect of radiation should have obliterated the vascular lesions to a considerable degree. This result is usually obtainable by dosage within the limit of tolerance of the adjacent normal tissues. Even though

the radiation in this case may be considered adequate, the beneficial effect was transient and there was insufficient change in the lesions to prevent recurrent bleeding. A higher dosage of intranasal radium, with less teleradiation, may have been more effective; however, at the time, additional radiation was thought inadvisable.

Moccasin snake venom therapy has since been started. It is too early for any comment on the ultimate effect.

DISCUSSION.

The diagnostic triad in hereditary telangiectasia consists of: *1.* bleeding, *2.* familial predisposition, and *3.* typical vascular lesions.

1. Bleeding: Hemorrhages usually begin early in life, even preceding the appearance of lesions. The amount and frequency are variable. In the great majority of cases epistaxis occurs, but bleeding may come from any part of the upper respiratory tract, the tongue, the gastrointestinal canal, the genitourinary system, the meninges, peritoneum or conjunctivae. It may follow slight trauma, as in coughing or sneezing, physical exertion, emotional stress, or even changes in humidity or barometric pressure. Death from hemorrhage rarely occurs, but repeated bleeding usually causes chronic debility and secondary anemia.

2. Familial Predisposition: The hereditary trait is not sex-linked, as in hemophilia, and the incidence is about equal in males and females.

3. Vascular Lesions: Due to a developmental defect, the thin-walled capillaries and venules are deficient in elastic connective tissue and undergo dilations, forming telangiectasia, nevi or angiomata. These dark red spots of various sizes fade on pressure. They are most numerous on the mucosa of the nose, mouth and lips, but also occur on other mucous membranes and on the skin.

In the differential diagnosis all possible causes for bleeding must be considered. The hemorrhagic dyscrasias are easily excluded by the absence of any abnormality of the bleeding. coagulation or clot retraction times. There is no increased fragility of the erythrocytes or decrease in the blood calcium.

Finally, the platelet, differential and white blood cell counts are within normal limits.

Reduction of the hemoglobin percentage and of the red blood cell count is usually present. The secondary anemia requires correction by diet, hematinics and blood transfusions.

Among other methods of treatment, the destruction of angiomata by cauterization is of value in stopping hemorrhage when the bleeding lesion is accessible. This may be accomplished by the use of chemical caustics, solid carbon dioxide, electrocoagulation or the actual cautery. O'Kane has recently advocated repeated submucous injections of quinine and urethane, 5 per cent solution, as an effective means of controlling epistaxis.[9]

Satisfactory clinical results, evaluated by the decrease in the severity and frequency of hemorrhages, have followed immunization with moccasin snake venom.[8] The mechanism in this therapy is presumed to be an increase in the capillary wall resistance. Its effect is universal on all the lesions. Despite a rather high incidence of reactions and the necessity for continued maintenance dosage, this method seems to have great merit and will probably become the treatment of choice.

SUMMARY.

1. A case is reported of hereditary hemorrhagic telangiectasia with recurrent epistaxis.

2. Radiation therapy was given to a total dosage of 300 mgm. hours intranasally, and 40,000 mgm. hours by teleradiation.

3. The beneficial result was transient. Further treatment with moccasin snake venom has been instituted.

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25 East Washington Street.

SPECIAL NOTICE.

At a Regular Meeting of the Council of the American Otological Society held on Nov. 26, 1938, at which all members of the Council were present — Dr. Newhart, Dr. Mosher, Dr. Fowler, Dr. Berry, Dr. Wishart, Dr. Harris and Dr. Friesner — the following resolution was duly made, seconded and unanimously carried:

"That the Council of the American Otological Society withhold from publication the paper entitled, "Rationale, Technique, Case Reports and Observations with Fistulization of the Osseous Labyrinth," by Dr. Samuel J. Kopetzky, Dr. Julius Lempert and Dr. Ralph Almour, New York, and that this resolution be published in THE LARYNGOSCOPE."

(Signed) DR. THOMAS J. HARRIS, Secretary.

DISCUSSION OF THE RELATION OF NASAL POLYPS TO THE KELOID.

DR. E. R. HARGETT, Springfield, Ohio.

The title is meant to call attention to the similarity of nasal polyps and keloids. A point to be brought out here is the question of just what is a polyp, in the strictest pathological sense? Boyd[1] classifies keloids under fibromata, and mentions soft fibromata in the mucous membranes of the mouth, bronchial tree and gastrointestinal tract, but does not mention under this classification fibroma of the nose. With such a common condition, and with its relative importance to the rhinologist, it is thought worth while to bring any new light possible to bear upon the problem.

Subal,[2] of Vienna, and others teach that injury, irritation and/or infection of the cornea over any appreciable length of time will produce a keloid of the cornea, plainly discernible in the slit lamp. Boyd[1a] states "the term polyposis must not be taken to indicate a pathological entity, for polypoid masses may be either neoplastic or inflammatory in nature." He also states[1b] "the mucous polypus of the nose bears no relation to a myxoma. Indeed, it is not a neoplasm at all, but a mass of inflammatory tissue, jelly-like in consistency, and containing mast cells and eosinophiles." He goes on to say[1c] "the lymphocyte, of which the mast cell is a form, appears to be an important, though frequently inadequate, means of defense against the spread of carcinoma, but here again the cells are probably histogenous rather than hematogenous in origin. . . The eosinophil is a mysterious cell which appears in considerable numbers, but for no apparent object, in very diverse conditions." Maximow[3] states "nothing is known of the functional significance of the mast cell." He says it is related to the basophil leukocyte, and describes it as having some lymphocytic characteristics.

Then, in addition, Boyd[1d] says "regeneration occurs even more readily in connective tissue than in epithelium. Shortly

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Sept. 23, 1938.

after injury these cells commence to proliferate. Division occurs, and the new cells put forth branching processes." Then,[1e] "a fibroma is a tumor composed of fibrous tissue." On the same page, "keloid is not a true tumor, but a tumor-like condition which generally originates in a scar. It consists of very dense fibrous tissue, the bundles of which run parallel to the surface." And then, in speaking of submucous fibroids of the uterus, "owing to the uterine contractions, the fibroid tends to become more and more polypoid, until it finally may be extruded into the vagina."[1f] Skillern[4] quotes numerous authors as to the nature and etiology of nasal polyps, and then states that the problem is not completely solved. He then states that the nasal polyp is a true nasofibroma. Uffenorde[5] states "polyps have no relation *per se* to infection," and so does not believe they have any infectious origin. Hajek[6] states that they develop most frequently in the middle meatus because of the "peculiar loose condition of the mucoperiosteal tissue covering the ethmoid bone." Grunwald[7] states that chronic infection necessarily precedes nasal polypi; Skillern[4] says that the polyp may follow as a result of it (chronic infection), and that particular portion of mucous membrane from an infection elsewhere in the nose, and not the infection of that mucous membrane itself.

Ewing[8] considers a nasal polyp a fibroma, and states that they are "probably always preceded by chronic rhinitis." He further says "here, as elsewhere, it is impossible to draw a sharp line between the products of chronic inflammation and tumor processes." He then states "many of them consist of nothing more than localized edematous areas of mucous membrane rendered protuberant by mechanical means, but without other changes." How can he tell that there has not been some increase in the number of connective tissue cells without complete serial sections of the whole area before and after the changes, which is physically impossible? Even this refinement of science cannot tell me for certain the exact count of the number of cells in a given area. And if there has been in these questionable areas any increase in fibrous connective tissue cells, then the protuberance is in the fibroma class. MacCallum[9] describes nasal polypi as true fibroma, and mentions nothing about them having any inflammatory origin; in fact, giving the impression that he believes that they have the same origin as any other neoplasm. He states, and pictures the

sections to show, that the only difference between a keloid and a nasal polyp is that in the latter the connective tissue cells are more sparsely arranged; however, he does not call attention to the similarity of the two, or actually compare them in any way by mentioning them together, other than that both are placed under the fibroma class tumor.

In Turner's[10a] new edition, Dr. Douglas Guthrie and Dr. Charles E. Scott devote a separate chapter to nasal polypus, and another and different chapter to new growths, evidently trying to separate the two as far as possible. They state that the nasal polypus "is in no sense a new growth." On the same page they say, "any condition of hyperemia or inflammation within the nose may give rise to polypus formation. Polypi may accompany . . . allergic conditions." The first quotation is one of the most dogmatic I have ever seen; the second is the most liberal. He then allows Mr. J. D. Lithgow[10b] in the same book to make the following statement, while speaking of the fibromata of the larynx: "These innocent tumors, or polypi, as the pedunculated variety are called, are either a degeneration or a new formation of the fibrous-tissue elements of the stratum propium of the mucosa. When pedunculated they somewhat resemble myxomata in appearance, and differ very little in structure from the nasal or aural polyp."

Lack[11] states "the production of nasal polyps is dependent on disease and irritation of the underlying bone, in the form of a rarefying osteitis or sclerosis." It is well known that bone in other parts of the body reacts to chronic infections and chronic irritation in the same way. So I believe that Lack has mistaken one of the pathological findings for the etiology of the polyp, when in fact the true etiological factor is overlooked. Woakes[12] made the observation that polyps are associated with "necrosing ethmoiditis, and underlying bone disturbances." With any severe infection or irritation in tissues so close to the bone, I would expect to find underlying bone disturbances, but would not attribute the whole pathological picture to this one finding.

I know that polyps are prone to return in many instances after most thorough removal by the best operators. When attention is called to it, one is struck by the similarity of this discussion to one of keloids. In fact, for all practical purposes,

they may be considered as identical. Both may develop following injury, irritation and/or infection, although in the nose the injury takes place rarely in physical form, in a manner crushing the more susceptible parts of the membrane, *i.e.*, those over the middle turbinate and ethmoids. But the chronic irritation, without infection, may be the result of repeated allergic attacks, inhalation of dust or chemicals, or as Skillern[7] states, an irritating discharge from another area. Some families and races are more liable to develop keloids, and also polyps, than others. The latest cancer research shows that abnormal growth stimulation takes place in normal cells exposed to the secretory products of injured cells, when the injury to the latter is prolonged, and not severe enough to cause the death of the latter. The mode of action is a natural one, beginning with the universal law of healing, "proliferation in the presence of injury." But this stimulation to mitosis from injured cells does not stop, as in clean wounds, but goes on and on. The neighborhood cells are chronically subject to injury not sufficient to cause their death, and thus the development of different cellular products, but continue to throw out that stimulant to mitosis in the same manner, giving the other cells in the vicinity no rest; and sometimes, finally, as in the case of the spirochete and the neosalvarsan, developing a generation of cell that has lost some of its former characteristics. In the case of the malignant neoplasm, then, the factor inhibiting mitosis is bred out. It seems probable that high voltage Roentgen therapy does not supply this inhibiting factor, but in a crude way subdues the mitotic stimulating factor. But if the high voltage Roentgen therapy is unfortunately regulated, the new type cell will, in accordance with Darwin's description, and again like the spirochete, develop a radioresistant cell. This fits in perfectly with the conditions producing polypoid proliferation in the nose, and is the logical explanation, the word injury being used in its broadest sense. Also, I see that, although the cancer research experiment referred to above clarifies and defines the reaction and result, surgeons and rhinologists have known for a long time that neither in, near nor around many keloids and nasal polyps do they see ulceration, slough, necrosis, granulations or other foreign body reactions. Thus, therefore, if the stimulation to new growth be local, as it certainly is, it must be from the secretions of the products of metabolic processes of injured, and not dead, cells.

It is impossible for the rhinologist to incise around the base of the polyp with a sharp knife, as the surgeon so carefully does the skin keloid, but he can follow his excellent example and recommend postoperative high voltage Roentgen therapy. It is one of the purposes of this paper to make that seem logical, and to encourage high voltage Roentgen after-treatment of nasal polypi. So the rhinologist must not indict himself too hastily when polyps are found in the nose several months after their supposed removal by him; as with keloids, the very trauma of the original removal may have instigated the return, especially in those with a family history of polyps. Or, as suggested above, he may have failed to correct some other source of irritation, even external, or infection in other parts of the nose, with discharge contacting the seat of the growths. Sound practice tells me, as in neurasthenia, to eliminate any possibility of actual pathological etiology before falling back on the easiest diagnosis.

All rhinologic writers on nasal polyps mention the terms hyperplasia or hypertrophy somewhere in the discussion, applying these terms to the masses. No one states clearly the fact that a polyp contains fibrous connective tissue cells other than those that existed in its basal membrane or starting point, and that these masses became such through the proliferation of these connective tissue cells, and their hyaline degeneration. Yet, all at one point or another imply it, admit its possibility, or make conflicting statements concerning it. Skillern's[7] statement, after confusing quotations and descriptions, that it is a true nasofibroma is the most definite of them all. Nothing drains from a polyp when it is punctured, incised or multiply incised, as from an edematous leg. It is a permanent structure unless removed surgically or by high voltage Roentgen therapy, as are other neoplasms. It can grow to such size as to be present in the external nares, or the pharynx, and exert such pressure as to enlarge the bony structure of the external nose, and all without any signs of any inflammation being present in the nose. And if it is inflammatory, where are the polymorphonuclear leukocytes, or the heavy lymphocytic infiltration, the granulation tissue, and other signs of chronic inflammation? The only logical conclusion is that the nasal fibroma is a soft fibroma, more comparable to the keloid than to any other entity. Both have a tendency to occur and recur in certain individuals, and not

in others. Thus, in the presence of chronic nasal infection, one person may develop a polyp, and another may not. It clears up the complaint of Ewing[8] that "it is impossible to draw a sharp line between the products of chronic inflammation and tumor processes." In the newer light of neoplastic development, how could anyone, and why should anyone try to draw such an artificial distinction, when there is none there? In support of my contention that polyps are tumors, I call attention to instances in which polyps have become malignant, as cited by Goldman[13] and others.

To sum up, it is certain that MacCallum[9] gives the best evidence on the subject for any jury, with his pictures of sections and exact histological cellular descriptions. The new experiments showing that cellular injury of any type can cause neoplastic proliferation explain other characteristics and occurrences of the polyp, and the fact that the polyp shows the recurring tendency of the keloid, an admitted fibroma, should definitely establish for anyone that the nasal polyp is a fibroma. In fact, this newer conception is the only basis upon which I can logically explain all the facts known about polyps, and the best basis upon which to surgically and therapeutically treat them.

CONCLUSIONS.

1. Nasal polyps are fibromas.

2. They have a keloid-like tendency to return after removal.

3. As with the keloid, this tendency to return can in some cases be prevented or partially prevented by high voltage Roentgen therapy.

4. Attention is directed to the cellular injury theory of the development of polyps, and the rhinologist urged to remove the source of this injury along with the polyp.

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First National Bank Building.

AMERICAN OTOLOGICAL SOCIETY, INC.

The Seventy-second Annual Meeting of the American Otological Society, Inc., will take place on May 22 and 23, 1939, at the Westchester Country Club, Rye, N. Y., under the presidency of Dr. Isidore Friesner.

The guest of honor of the Society will be Prof. Otto Marburg, who has chosen for his subject, "Modern Views Regarding the Anatomy and Physiology of the Vestibular Tracts." A varied program of great scientific interest is in preparation.

The following candidates for active membership were approved by the Council at its meeting, Nov. 26: Dr. Daniel S. Cunning, New York; Dr. Joseph G. Druss, New York; Dr. Thomas C. Galloway, Evanston, Ill.; Dr. Westley M. Hunt, New York; Dr. Frank J. Novak, Chicago; Dr. Otto M. Rott, Spokane, and Dr. Henry L. Williams, Rochester, Minn.

The membership of the Society is: Honorary, 12; Senior, 28, and Active, 107.

SALIVARY CALCULI.

DR. LOUIS R. EFFLER, Toledo.

Introduction: Two interesting cases of stones in the salivary ducts have come to our attention in the past year. Since such cases are relatively uncommon in private practice, we shall relate briefly their history in particular, and add a few remarks on the behavior of such stones in general.

CASE REPORTS.

Case 1: Mr. M., age 45 years. History: Had been troubled for the past eight years with a peculiarly acting swelling in the left neck. This swelling was never entirely absent during this whole period; it was, indeed, for varying periods of time quite small and innocent in appearance. For the most part, however, it seemed progressively on the increase in size and, especially in late years, more consistently troublesome. In general, during its quiescent state, it had increased in size from that of a small marble to that of a good sized English walnut.

Particularly in the last year or two this tumor gave trouble around meal times. Attempts at eating were frequently, though not always, accompanied by marked swelling of the whole left neck. Meals had often to be cut short or entirely eschewed, due to this swelling, with its accompanying pain.

All sorts of local applications had been applied during these periods of heightened pain and swelling, and had seemed, until recently, successful in coping with the situation. Repeatedly, just about the time that our patient had felt he needed medical attention, the whole matter would seem to clear up spontaneously.

Physical Examination: When first seen by us on Oct. 1, 1937, at a time when this swelling had been more than usually protracted, the whole left neck was involved. The swelling was generalized and looked like an ordinary case of cervical adenitis, acute, nonsuppurative, left. The whole chain of tender lymph glands could be palpated, in addition to which we thought we could outline a greatly hypertrophied submaxillary gland at the angle of the left jaw.

The patient was in evident distress. He was admitted to the hospital for a few days' observation, with orders to abstain from all food for 24 hours. At the end of this time the swelling had subsided considerably.

For purposes of experiment, we watched him take his first meal after his 24-hour starvation period. Almost immediately, his left cervical swelling recurred. Quite contrary to expectations, this swelling was more generalized than localized in the region of the submaxillary gland.

Our tentative diagnosis of stone in the left submaxillary gland or duct was sufficiently sustained to warrant the taking of an X-ray.

This X-ray revealed the presence of a small round stone (see Fig. 1).

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Sept. 15, 1938.

Operation: Since palpation failed to reveal the exact locality of the stone, either within the mouth or in the left neck, we concluded the stone must be located in the hilus of the gland itself. An open operation was performed, in which the submaxillary salivary gland was removed en masse. The stone was recovered approximately at its suspected site.

Sequelae: Quite to our surprise, this operation succeeded in accomplishing two things: 1. It cured completely his distress at eating and made, in fact, eating again a pleasure. 2. It removed a distressing "focal infection," from which the patient had been suffering for years. In fact, he had been treated consistently but unsuccessfully for both a "stomach ulcer" and a "chronic prostatitis." The former had been accompanied by a distressing hyperacidity, which not even a careful diet had relieved;

Fig. 1. Case 1. Showing X-ray of stone nicely outlined at angle of left jaw. It was pea-sized and pea-shaped, and caused blockage of the left submaxillary duct. Symptoms were first noticed eight years before operative removal (see text).

and the latter had been accompanied by a painful micturition and the necessity for three or four night-risings regularly. Strange to say, both of these symptom-complexes completely and miraculously disappeared within one week after the operative removal of his salivary calculus!

Case 2: Mr. E., age 25 years. History: Had been troubled for the past six years with the same type of swelling in his left neck, and a peculiar swelling, in addition, under the left tongue. On questioning, he could not be certain that these swellings were always associated with the intake of food. At least, he was not so sure about the beginning of his trouble, though he was quite sure on this point with regard to the past few weeks. Increasingly painful deglutition at meal times was becoming so severe that quick relief was sorely needed. The patient had already lost 10 pounds in weight from enforced semistarvation.

N.B.: On his history alone, the diagnosis of suspected salivary calculus was made by our office nurse. We are particularly happy to give her the credit for her alertness and smart questioning. At the same time, we are indirectly complimenting ourselves and taking some credit for her proper training. The behavior of these calculi is such that they should seldom be overlooked, provided the symptoms are reasonably clear-cut.

Physical Examination: Inspection of the left neck revealed the same type of generalized swelling as noted in our first case; in addition, there was noted a moderate sized and slightly reddened swelling under the left tongue, which pushed the tongue up noticeably from the floor of the mouth.

A test meal, before our eyes, resulted in a marked increase in both these swellings. In fact, the swelling beneath the left tongue increased so markedly and became evidently so painful as to embarrass swallowing.

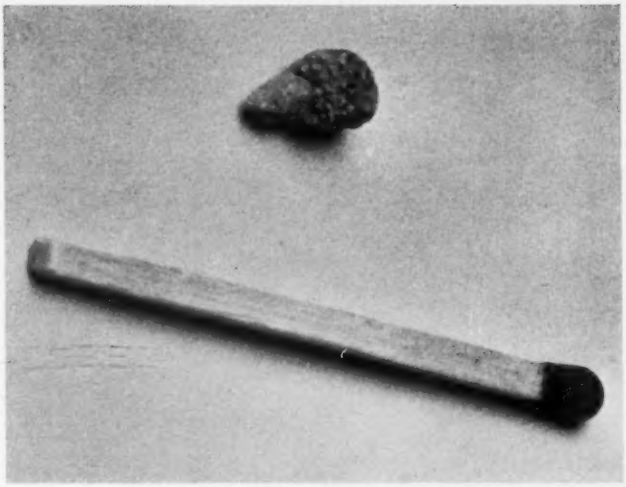


Fig. 2. Case 2. Showing picture of calculus compared to ordinary parlor match, indicating the relative size. It was conical-shaped, like a miniature chicken croquette, and of rough surface, and caused blockage of one of the left sublingual ducts. Symptoms were first noticed six years before operative removal. The peculiar shape of this stone would indicate that its base lay in the hilus of the gland, while its apex extended into the gland duct (see text).

Tentative Diagnosis: Attention was directed mainly to the swelling beneath the left tongue. The presence of this reddened tumor mass, which with the attempt at taking food had increased to the size of a small lemon, made a tentative diagnosis likely of a stone in the left sublingual gland or duct.

We dispensed with the formality of an X-ray picture at this juncture, because we felt that incision and drainage of an apparent sublingual abscess, left, was first indicated.

Operation: Incision and drainage of this tumor mass recovered about two ounces of glairy mucopus. With the right hand on the outside of the

neck for counter-pressure, the fingers of the left hand were introduced into the mouth beneath the left tongue. Combined palpation and "stripping" toward the wound were rewarded with the sudden popping out into the wound of a small cone-shaped calculus (see Fig. 2).

To account for its peculiar shape, we suppose that its broadened base began in the hilus of the gland and that its narrowed apex extended into the narrow mouth of one of the gland's numerous ducts. To account for its recovery, we suppose that the accumulation of pus and salivary contents distended the duct sufficiently so that, instead of remaining wedged within the duct, it practically "floated" out into the wound, together with the abscess contents.

Sequelae: Within 48 hours the patient was eating once more and swallowing with reasonable comfort. After one week all signs of trouble under the left tongue had completely disappeared. The left submaxillary gland, however, still remains hard and enlarged, though not tender. A 10-pound weight gain in this short space of time compensated quickly for the above-mentioned wasting from enforced semistarvation.

Brief Review of Anatomy of Salivary Gland System: For purposes of review, it must be remembered that there are three sets or pairs of salivary glands, with their respective ducts.

a. The *parotid glands* lie in front of the ears on each side. The opening of these glands for the conveyance of saliva into the mouth is by way of right and left "ducts of Stenson." Each duct is about two inches long and one-eighth inch in diameter. Each opens into the mouth atop a well marked papilla that lies in close relation to the upper second molar tooth on each side.

b. The submaxillary glands lie under the angle of the jaw on each side. The opening of these glands into the mouth is by way of right and left "ducts of Wharton." Each duct is about two inches long and opens into the floor of the mouth on either side of the frenum of the tongue. Again, each orifice is perched atop a little nipple-shaped papilla.

c. The *sublingual glands* lie, respectively, under right and left lateral folds that run beneath the tongue on a line parallel with and just inside the lower teeth on either side. Unlike the parotid and the submaxillary glands, which have a *single* duct for each, the sublingual glands have *multiple* ducts, to the number of about 15 or 20 for each. These multiple ducts are called the "ducts of Rivinus." Each minute duct has a corresponding minute duct opening on a minute papilla, the whole series of which empty along the course of the above-mentioned lateral folds.

Brief Review of Surgical Pathology of Salivary Gland System: Each salivary gland has an interest from a pathological standpoint.

a. The *parotid glands* are known to have calculi, though not so commonly as the submaxillary and the sublingual gland systems. The parotids are the site both of a contagious form of "mumps" and a noncontagious form of so-called "surgical mumps." The latter follows occasionally as a postoperative complication of abdominal surgery. It may be due indirectly to dirty mouths, in which the ever-present bacteria take advantage of the patient's lowered resistance.

b. The *submaxillary glands* are, pathologically speaking, best suited to the formation of stones or calculi. The right concentration of salts in the saliva of this gland forms concretions, just as in the kidney or gall bladder. If these salts are deposited only on the teeth, we speak of the deposit as *tartar.*

A calculus in a submaxillary gland or its duct may be a long time in forming. The stone may be lodged in the gland substance itself, its hilus or anywhere in the course of its two-inch long duct. Strangely, the blockage is *not continuous.* If it were, relief would have to be forthcoming early. As it happens, however, symptoms are directed to the affected gland *intermittently.*

When a patient eats, the gland is in a state of greatest activity. Because the outlet for its secretions is wholly or only partially blocked, the back-flow causes both the gland and the adjacent neck structures to become greatly swollen and painful. This painful swelling naturally causes the patient to stop eating. Between meals the swelling subsides—only to start up again at the next meal time from a mixture of taste, smell or even sight of food.

This state of affairs may go on for years. Occasionally, an inflammatory process causes all the symptoms to become acutely active; but just about the time surgical interference seems indicated the stone may shift its position and bring about spontaneous drainage, with relief of symptoms for this particular attack.

In general, however, relief is only short-lived. The stone grows slowly larger, so that symptoms of blockage become definitely more frequent, more painful and more urgently necessary to relieve. The stone may grow as large as a bean or even, at times, as large as a walnut. Throughout its growth, however, it must be remembered that *size* alone does not count so much as *position* of the stone.

c. The *sublingual glands* are attacked rather infrequently by a sort of retention cyst, called a "ranula." This swelling is located underneath the tongue on one side or the other. Due to the multiplicity of duct openings, it is easy to understand how one or more of these openings may become plugged by some low-grade mouth infection.

Pathologically, the sublingual gland system may also contain calculi or stones. The same general remarks addressed above to the submaxillaries may apply to the sublinguals. The only difference is the site of the swelling; neck in the former, and sublingual region in the latter. The sublingual swelling increases and decreases in size with a calculus; while it increases only or remains stationary with a ranula. In either case, it may be subject to intercurrent infection and become acutely inflamed.

The contents of a ranula are saliva and mucus; the same is true of a retention cyst from calculus, unless it becomes infected with pus. The ranula must receive expert surgical attention, usually external incision and dissection, to prevent recurrence; the sublingual retention cyst from calculus must aim at the removal of the calculus to prevent recurrence. Neither will cure itself spontaneously, but requires surgical interference.

SUMMARY.

1. Salivary calculi are not uncommon.

2. They are more common in the submaxillaries and the sublinguals than in the parotids.

3. The history of calculi in these gland systems is noted for its chronicity, its periodic relation to the ingestion of food, and its intermittent rather than continuous character.

4. The size of the stone seems to be of less importance than its position.

5. Partial blockage of the duct involved is more common than complete blockage, except in the later stages.

6. The submaxillary calculus gives signs of swelling in the neck of the affected side.

7. The sublingual calculus gives signs of swelling in the sublingual region of the affected side.

8. All signs of swelling are definitely increased with the ingestion of food.

9. The removal of submaxillary calculi may have to be accomplished by an external neck incision, although occasionally they may be recovered through the oral route.

10. The removal of sublingual calculi is, almost without exception, accomplished through the oral route.

222 Michigan Street.

MISSISSIPPI VALLEY MEDICAL SOCIETY

1939 ESSAY AWARD.

The Mississippi Valley Medical Society offers a cash prize of $100.00, a gold medal and a certificate of award for the best unpublished essay on a subject of interest and practical value to the general practitioner of medicine. Entrants must be members of the American Medical Association. The winner will be invited to present his contribution before the next annual meeting of the Mississippi Valley Medical Society at Burlington, Iowa, Sept. 27, 28 and 29, 1939, the Society reserving the exclusive right to first publish the essay in its official publication — the Mississippi Valley Medical Journal (incorporating the Radiologic Review). All contributions *must not* exceed 5,000 words, be typewritten in English in manuscript form, submitted in five copies, and must be received *not* later than May 1, 1939. Further details may be secured from Dr. Harold Swanberg, Secretary, Mississippi Valley Medical Society, 209-224 W. C. U. building, Quincy, Ill.

THE EFFECT OF SULPHYDRYL COMPOUNDS IN OTO-LARYNGOLOGY.*

DR. WILLIAM MCPHERSON FITZHUGH, JR., San Francisco.

Introduction: There is no denying the fact that living as well as dead matter is made up of molecules, atoms, ions and electrons, and the control of biological forces is unceasingly accomplished by chemical and physical methods. In studying the growth phenomena everyone should realize that it is only one of an array of bewildering processes and not an isolated reaction; also, in the last few years the chemical as well as the physical factors have been taken into consideration. Hammett and Reimann in their cancer research decided to approach the question by first determining what chemical compound was essential for normal cell development and then if lack of, or slight modification of, said compound would be the cause of, or retard, neoplastic proliferation. Atrophy is the opposite of growth; therefore, I thought it would be extremely interesting to know if the lack of the chemical compound necessary for growth could be the cause of atrophy. The author was also interested to find its value as a medicant in other pathological conditions.

HISTORY.

All living organisms proliferate; therefore, any one of them could be used to find out the chemistry of cell growth. Hammett chose plant roots and lead as a reagent because it is an active chemical substance, readily identified, and it is also interferes with cell growth. The roots were suspended in a lead nitrate solution, and it was found that the lead concentrated in that part of the root where cell division is most active. It was precipitated in the nuclei during mitosis, and after months of painstaking analysis the lead was found to be combined with some reduced organic sulphur radical, thus

*Thesis submitted to the faculty of the Graduate School of Medicine of the University of Pennsylvania, in partial fulfillment of the requirements for the Degree of Doctor of Medical Science — D.Sc.(Med.) — for graduate work in otolaryngology.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Oct. 22, 1938.

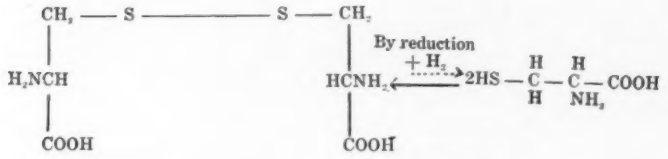
confirming the work of previous investigators who found high concentrations of sulphydryl in regions of mitosis. Offering sulphydryl in proper concentration to growing root tips, Hammett found an increase in the length of the roots compared to the control. He also demonstrated that this amplification was due to an increase in cell division and not an enlargement of the cells themselves.

Other investigators found that in nontoxic concentrations of sulphydryl there was an increased rate of proliferation with the paramecium, ameba, hydroza, worms, arthropods and mollusks.

Hammett and Reimann then removed the skin from over the shoulder blades of rats; to the right side was applied a sulphydryl compound and to the left its chemical control. After constant application for four days and nights it was found that the side to which sulphydryl was applied was completely healed, while the other side had but started in the usual way. After the rat experiments were completed, Hammett and Reimann decided to try sulphydryl on man. The first case was an elderly man whose thigh had been shattered by the kick of a mule. Complicating the trauma was a large varicose ulcer on the same leg; naturally, it was impossible to consider the use of a cast or any other proper dressing to the injured femur without first healing the varicose ulcer; so, with the permission of the surgeon in charge, they were allowed to apply a nontoxic concentration of thioglucose, and at the end of the 24 hours of constant wet applications the dressings were removed and 2 inches of skin had grown around the edges of the ulcer toward the centre; continuing its application, the lesion completely healed. Since this time surgeons and physicians at the Lankenau Hospital and other institutions have applied sulphydryl compounds to various ulcerations and wounds with marked diminution in the time of healing, but it has not been efficacious in purulent cases. The sulphydryl group is extremely labile, which is natural if it plays an important part in proliferation; and it is evident that the purulent exudate was instrumental in changing the compounds containing the sulphydryl radical; therefore, experiment must be done with due regards to the peculiarity of their chemical behavior.

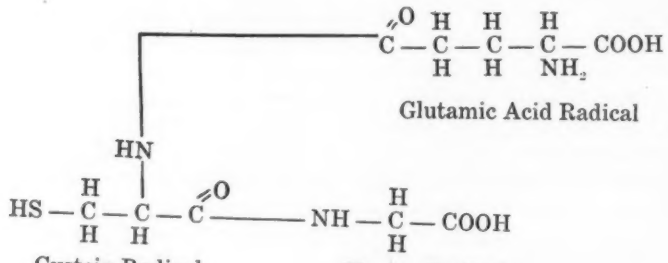
CHEMISTRY AND TECHNIQUE.

There are only a few sulphur compounds in the food we eat which can be utilized by the animal organism; and their metabolic end-products are the sulphates. The chief sources of sulphur are the amino acids, cystine, methionine and ergothionine.



It was found that in most experiments with animals a cystein which is essential for cell proliferation is exceedingly unstable, easily oxidizes completely to cystine, and, therefore, difficult to procure commercially. Methionine and ergothionine, also, are not manufactured commercially.

Cystein, combined with glutamic acid, forms glutathione, which has been found in the body.



GLUTATHIONE

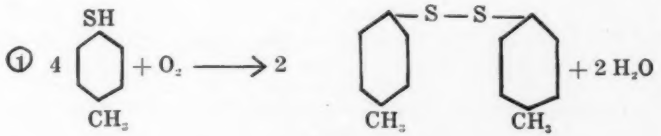
Hopkins has shown that washed muscle tissue yields a thermostable component which, when mixed with glutathione, forms a system capable of absorbing atmospheric oxygen. The glutathione contains a sulphydryl radical, although fairly stable under certain conditions (such as the above) it will finally oxidize by joining an SH radical of another molecule.

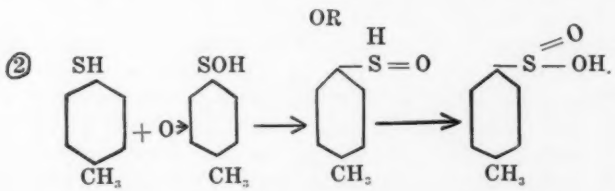
Glutathione has been used successfully for animal experimentation but is too expensive to manufacture commercially for medical application.

The problem, then, was to find some sulphydryl compound which was comparatively inexpensive, could be easily procured, and was not too unstable. Thioglucose* was used at first with marked success but, in many cases, the glucose acted as a good media for bacterial growth. A change was then considered to obtain a compound which would be bacteriocidal and, at the same time, would do very little damage to cell proliferation; so, after trying out numerous compounds, p-thiocresol was found to be the best drug available.

p-Thiocresol

Parathiocresol will in alkali media very soon oxidize to a variety of compounds.





There are many steps of partial oxidization which, at present, are of very little interest to the clinician.

*I was unable to find out the exact isomer of thioglucose; therefore, I have neglected to include any formula.

Hammett and Reimann found that the continued application of sulphydryl did not give the tissue enough time for cell differentiation and organization to take place. They decided, therefore, to stop the sulphydryl every four days and apply for 24 hours normal saline only. Parathiocresol is insoluble in water; therefore, it has to be dissolved in five times its weight of alcohol. This solution is then added to water and a fine emulsion results. All aqueous emulsions have to be made fresh, as, in the case of p-thiocresol, complete oxidization takes place in 24 hours. Distilled water should be used because sulphydryl compounds in the presence of copper, which comes from most plumbing, will immediately cause its oxidization.

ACTION OF PARATHIOCRESOL ON CILIATED EPITHELIUM.

The author thought it was necessary before arriving at the therapeutic value of p-thiocresol for pathological conditions of ciliated epithelium to note, first, its effect, if any, upon ciliated action. The esophagus of a frog was extirpated and pinned extended upon a cork. The criterion of the effect of the p-thiocresol was the time, compared to the time of the control, required for the mustard seed to travel 1 cm. One-half of the esophagus was bathed in p-thiocresol solution, while the other half was treated with a solution similar to the p-thiocresol itself except for the presence of the reagent. Readings of each esophagus were begun within two minutes of the time the frog was killed and were continued for five minutes. Ordinarily four to six readings were taken.

Parathiocresol is insoluble in water, and it was found impossible to make satisfactory emulsions other than by dissolving it first in alcohol and then diluting the alcoholic solution. Solutions containing one part of the drug in 99 parts of alcohol were found to be best and gave satisfactory emulsions on dilutions with water to all concentrations between 1/500 and 1/100,000. More highly concentrated solutions in alcohol, made with the object of reducing the concentration of alcohol in the emulsions used for the test and in the control solutions, precipitated crystals of p-thiocresol on dilution.

Attempts were made to replace alcohol with glycerine and with propylene glycol but without success, because of the appearance of large crystals of p-thiocresol after dilution.

Diethyl ether and diethylene oxide, while good from the point of view of the character of the emulsion formed, were unsatisfactory because of the known toxicity of the solvents.

Two series of experiments were carried out, the first to compare solutions of p-thiocresol in alcohol with solutions of alcohol of the same concentrations, and the second to compare solutions with p-thiocresol in alcohol with solutions of p-cresol in alcohol. A number of frogs' gullets were treated with the solutions of each concentration, and the following statistics were calculated according to the method of R. A. Fisher:

$$\bar{x} = \frac{s(x)}{n^1}$$

$$\frac{s^2}{n^1} = \frac{s(x - \bar{x})^2}{n^1(n^1 - 1)}$$

$$T = \bar{x} \div \sqrt{\frac{s^2}{n^1}}$$

$$n = n^1 - 1$$

where the symbols have the following meanings:

n^1 = the number of frogs.

x = the difference in the times between test and control for one frog.

$\bar{x}$ = mean of the differences for n^1 frogs.

$s(x)$ = summation of x for n^1 frogs.

$\frac{s^2}{n^1}$ = the variance of the difference between the means for n^1 frogs.

T = the mean of the differences divided by the standard deviation of the difference.

The values of the mean difference, $\bar{x}$, the variance, $\frac{s^2}{n^1}$, and T are given the following two tables of results. The probability, P, that $\bar{x}$ differs significantly from zero by chance, was taken from Fisher's tables, p. 166.

For the series of p-thiocresol against alcohol, only at concentration 1/1,000 is it certain that the thiocresol had a stimulating effect on the action of the ciliated epithelium. The probability that the observed acceleration of the other concentrations could be obtained by chance is too great to warrant a valid conclusion that p-thiocresol stimulates the cilia. There is, however, no evidence that p-thiocresol inhibits the action of the cilia.

No. of Frogs, n^1	Test Solution		Control Solution						
	P-thiocresol	% Alc.	Sec., AV.	% Alc.	Sec., AV.	$\bar{x}$	$\frac{s^2}{n^1}$	T	P
2	1/500	14.0	∞	15.0	∞	—	—	—	—
7	1/1,000	7.0	25	7.5	34	9	10.2	2.8	0.03
3	1/5,000	1.4	24	1.5	25	1	1.33	0.87	0.47
4	1/10,000	0.7	18	0.75	22	4	8.5	1.27	0.3
4	1/20,000	0.35	25	0.37	32	7	40.5	1.1	0.35
3	1/50,000	0.14	14	0.15	21	7	9.0	2.3	0.15
4	1/100,000	0.07	21	0.07	27	6	52.0	0.8	0.48

The second series, p-thiocresol against p-cresol, both dissolved in alcoholic solution, is more definite. At only one concentration, 1/50,000, is the action of the drug uncertain. At all other concentrations the results show that p-thiocresol, compared to p-cresol, has an accelerating effect.

No. of Frogs, n^1	Test Solution			Control Solution			$\bar{x}$	$\frac{s^2}{n^1}$	T	P
	P-thiocresol	% Alc.	Sec. AV.	P-cresol	% Alc.	Sec. AV.				
1	1/500	14.0	∞	1/500	15.0	∞	—	—	—	—
4	1/1,000	7.0	27	1/1,000	7.5	45	18	18.9	4.2	0.03
7	1/5,000	1.4	16	1/5,000	1.5	22	6	4.6	2.6	0.04
6	1/10,000	0.7	24	1/10,000	0.75	28	4	1.8	3.2	0.03
5	1/20,000	0.35	14	1/20,000	0.37	18	4	1.9	2.7	0.06
3	1/50,000	0.14	20	1/50,000	0.15	21	1	0.94	1.2	0.35

CONCLUSIONS.

1. There is no evidence that p-thiocresol in therapeutic concentrations inhibits ciliary action.

2. In the second group of experiments evidence shows that p-thiocresol is stimulating.

PARATHIOCRESOL IN ATROPHIC RHINITIS.

Sulphydryl compounds have been found by previous investigators to stimulate cell reproduction. In atrophic rhinitis there is a degeneration of the ciliated epithelium and mucous glands with no replacement; and, therefore, it occurred to me that, in all probability, this degeneration and lack of reproduction was due to the absence of some growth-stimulating substance; and, owing to the success of Hammett and Reimann, I decided to use p-thiocresol. May I say here no attempt was made by the author to differentiate the various etiological factors which caused, or were causing, this pathological condition? Knowing, however, through the experience of previous investigators, that sulphydryl compounds were readily oxidized (losing their growth-stimulating properties) in the presence of purulent exudate, all cases complicated with chronic purulent sinusitis were excluded from this series. In order to apply p-thiocresol to the mucous membranes of the nose a suitable water-soluble base was needed whose viscosity was such that it would remain for some time in the nose; and, at the suggestion of the McNeil Laboratories, a tragacanth jelly was found to be the most suitable.

Glycerin	6 ozs.
Water	18 ozs.
Mineral oil	1½ ozs.
Powd. tragacanth	240 grs.
Oil of pine	120 min.

The question now arose as to what strength of p-thiocresol to use which would be the most beneficial without being too toxic to the cells; and, after taking into consideration the results found with the ciliated epithelium of a frog and, also, with the advice of Dr. Stanley Reimann, we decided to use 1:1,000. Sulphydryl compounds are notoriously unpleasant to olfaction; and, therefore, a trace of either oil of pine or oil of rose is used as a deodorant. This prescription is placed in four-ounce tubes. The patients use the following schedule: Every morning the nose is douched with warm saline, which helps remove any crusts that might have formed during the night. They are taught to place the end of the tube in the nostril while the head is extended (so that a line drawn from the point of the chin to the external auditory meatus is

perpendicular to the floor). After squeezing some of the solution, the outside of the nostril is gently massaged with the finger. The patients use the p-thiocresol three or four times a day (after meals and before going to bed) for four days. On the fifth day they rest, allowing time for cell differentiation to take place.

In order to find out whether our results were due to the p-thiocresol, five cases which were benefiting from the use of the drug were unsuspectingly given tubes of plain tragacanth containing onl ythe deodorant. All of them said that the jelly was not having the same effect that it had before. This led the author to believe that the p-thiocresol was an important adjunct to the prescription.

An attempt was made in one case that had used the p-thiocresol for six weeks to remove a small section of the mucous membrane from the inferior turbinate for biopsy, and on microscopic examination it was found to be ciliated epithelium. However, nothing was proved because no section had been taken prior to treatment. The author was afraid to attempt to confirm these findings. The following is a resumé of 16 cases:

Case 1: P. S., age 17 years, white, school girl. Complained of nasal obstruction, crusts and dryness of throat for three years, which were getting progressively worse. At first, in the hospital crusts had to be removed manually and by irrigation. Later, under p-thiocresol treatment, the crusts disappeared and there appeared to be a bilateral proliferation of the mucous membrane of both sides of the septum. The inferior turbinates were gradually increasing in size. After four months of successful treatment the patient moved and was not seen again.

Case 2: H. C., age 12 years, white, school boy. Complained of crusts, ozena, dryness of the nose and clearing the throat with a hawking cough. Prior to this, patient was exposed for four years to tuberculosis from his mother, which resulted in her death. Patient had pneumonia twice when a baby. He also had two attacks of pleurisy. There was a question that the patient may have tuberculosis and was, therefore, referred to the chest clinic for further study. Patient treated for 25 months with p-thiocresol. Has some return of color in the mucous membranes with increase in the mass of the turbi-

nates. There is still some crusting between both middle turbinates, but it is much less and can be easily removed.

Case 3: M. G., age 39 years, white, female, stenographer. Had suffered from anosmia and crusts for 12 years following scarlet fever. Had been to 11 doctors, one of which operated by raising floor of nose, with only temporary results. Patient had used p-thiocresol jelly for two months, and there is a general increase in the soft tissues of the nose with a diminution of the crusts. Patient feels more comfortable and she says it is the best local treatment so far.

Case 4: B. S., age 13 years, white, school boy. Patient had complained of ozena for several years, which had recently become progressively worse. Belonging to a Christian Science family, the child had received no medical attention. The mother's faith (probably induced by the odor) had weakened and he was brought to the clinic. Examination showed crusts and atrophic structures in both nares. The patient after two months of p-thiocresol showed marked improvement — there was no odor, the crusts had disappeared and the inferior turbinates were enlarging. Five months after starting treatment the p-thiocresol was discontinued. Observation over a period of 15 weeks showed no recurrence.

Case 5: D. R., age 31 years, white, female, stenographer. Complained of losing her job because of the odor of her breath. Examination showed marked atrophy of the right nares, filled with crusts, while the left was only moderately affected. After p-thiocresol therapy patient was free from all complaints in two weeks. Prolonging the periods between treatments appeared to have no disturbing effects — when I last saw the patient she was using it once a week.

Case 6: R. M., age 22 years, white, female, clerk. Complaints: crusts and bad breath. Patient said that two sisters had the same trouble. All previous medical treatment, although beneficial for a while, was of no avail. One year after starting p-thiocresol treatment patient showed marked improvement and was well satisfied.

Case 7: L. M., age 29 years, white, housewife; sister of R. M. Had usual complaints. Patient had plastic which helped for a while. Could not take iodine in any form because of sensitivity. Under p-thiocresol therapy the crusts and ozena

disappeared, and the patient was still continuing its application but the intervals of rest were longer.

Case 8: W. G., age 42 years, black, female cook. Three months previously patient had an attack of influenza which left her very weak. For the last three weeks the back of her nose felt dry, she had a lump in her throat, and every morning she blew out some crusts from her nose. The people she was working for objected. Two months with p-thiocresol treatment showed marked improvement. There was no longer any crusts and symptoms had disappeared. Two months after stopping treatment patient had no recurrence of symptoms.

Case 9: M. D., age 18 years, school girl. Complains of blowing crusts from the nose for three years, which have become progressively worse. Three years ago her family began to complain of the foul odor, and from actions of strangers when she visited public places she felt she was avoided on that account. She has a sister who also has the same complaints. Examination showed a marked atrophic rhinitis. On using p-thiocresol she gradually lost the foul odor and regained her sense of smell. Six months later we found only small crusts under both middle turbinates, and the mucous membrane is pink, while the turbinates are somewhat larger. She claims great relief and has more comfort than ever before.

Case 10: J. R., age 19 years, housewife, the sister of M. D., suffered from the same complaints. Blood pressure was 100/70. Rather poorly developed. Extremities disproportionately thin. Lack of hair in the axilla but normal amount of pubic hair. Diagnosed by endocrinologist as hypofunction of the adrenals. Examination of her nose showed markedly deviated septum to the left with atrophy of the soft tissues and plenty of crust on the right naris. On using p-thiocresol the symptoms were greatly relieved six months later, but she used the drug sparingly.

Case 11: J. S., age 15 years, white, school girl. Patient had had dryness of the nose and throat for several months. Examination showed typical atrophy of the turbinates with crusts. After using p-thiocresol for eight weeks the patient's symptoms disappeared, and two months later she discontinued coming to the clinic.

Case 12: R. N., age 44 years, black, male, plumber. Patient came to the clinic complaining of the fact that the family objected to the odor of his breath. Had anosmia for 18 months and dryness in back of throat. Had crusts in nose, which were expelled with a great deal of difficulty. After three weeks' treatment of p-thiocresol patient showed some improvement, in that crusts were diminished; however, patient grew tired and stopped therapy against the advice of the doctor.

Case 13: Age 11 years, white, school boy. Has had crusts in the nose for five months following an attack of measles. Patient used p-thiocresol for one month. The crusts diminished and there was a thickening of the turbinates and the anterior part of the septum. The author believes that this was an early case of atrophic rhinitis.

Case 14: G. M., age 33 years, yellow, male, clerk. Has been sickly nearly his whole life. Examination showed a roomy nose with plenty of crusts, which responded to p-thiocresol therapy. Six months after starting treatment patient died from pneumonia. Autopsy not granted.

Case 15: F. S., age 15 years, white, school boy. Patient noticed for several months that his nose felt very dry and he had lost his sense of taste. On examination patient had crusts, associated with anosmia. Parathiocresol therapy appeared to be beneficial for a while, and then suddenly had no effect. Under other therapy patient's condition is improving.

Case 16: S. O., age 30 years, white, female, chocolate coater. Crusts in nose, intermittent attacks of nausea early in morning, dryness of the throat — sometimes a burning feeling — for 20 years. Patient says that she was anemic at age 8 years. Four years ago floor of nose raised with ivory. This and all local application appeared to have only temporary effect. Parathiocresol therapy increased the size of the inferior turbinates and diminished all symptoms. Condition became stationary and treatment was, therefore, changed.

DISCUSSION.

Analysis of my series of 16 cases shows only two who were not permanently benefited by the local application of p-thiocresol jelly. Four of the cases were cured, and of the remaining 10 only three, for various selfish motives, discontinued using the drug.

It is true that some of these cases may not have had classical atrophic rhinitis; yet all of them had typical signs and symptoms and the diagnosis was collaborated by two or more rhinologists. My observations were only carried over a period of 2½ years, which is hardly conclusive, because those cases which at present appear completely cured or benefited by the use of the drug may only be having a recession of symptoms.

Granting that atrophic rhinitis is a systemic disease with local manifestation in the nose, I believe that the use of sulphydryl compounds for topical application is the best approach so far instituted, because they tend to supply to the ciliated cells that chemical substance necessary for their continued well-being.

At present most authorities agree that the degeneration of the nasal mucous membrane and mucous glands is due to some endocrine dyscrasia — hyposecretion of the adrenal cortex — which, I believe, probably acts in some way to diminish certain intracellular compounds. In order to insure final success in the treatment of atrophic rhinitis, the local application of sulphydryl compounds should be combined with some general therapy. This question is beyond the scope of this paper.

PARATHIOCRESOL IN ATROPHIC DRUMS.

Atrophy of the tympanic membrane is the sequela of a large central perforation associated with local and general lowering resistance of the patient. On examination the membrana tympani appears to have the consistency of tissue paper and, in some cases, using a Seigle otoscope shows marked relaxation. Histological examination shows that this opening in the drum has been covered by the squamous epithelium and mucous membrane, but that there is lack of generation of the fibrous tissue layer between them. The patients usually complain of flapping in the ear during conversation and respiration. The author thought that, if the p-thiocresol would thicken the squamous epithelium when applied to the external surface, it would, in all probability, have the same effect on the lateral covering of the drum. Parathiocresol, 1:1,000 in tragacanth jelly, is given to the patient to be applied four times a day for four days, and on the fifth day rest is instituted so that cell differentiation can take place. The patient is seen three times a week by the attending physician, who carefully wipes

the walls of the external auditory meatus with a 0.5 per cent sodium hydroxide solution in order to neutralize any p-thiocresol on its surface, which prevents the squamous epithelium of the auditory meatus from proliferating and resulting in atresia of the canal. The author wishes to report the following four cases:

Case 1: R. L., age 38 years, white, male, school teacher. Complains of a sensation of flapping in the ears for the last seven months. Two years ago patient had right acute, purulent otitis media with spontaneous rupture. He gave a history of intermittent otorrhea which lasted for over a year — in fact, patient said last discharge took place nine months ago. Examination of the right ear shows healed heart-shaped perforation which is thin and moves in and out during conversation. Following p-thiocresol therapy for three months the tympanic membrane was dull, slightly opaque, and hearing was not diminished. Patient says that he no longer has the flapping sensation and appears greatly relieved. Advised to use p-thiocresol once a month for four days.

Case 2: J. B., age 29 years, white, female, stenographer. Came to the clinic complaining of a peculiar sensation in the left ear of five months' duration. She gave a history of an attack of bilateral acute suppurative otitis media complicating scarlet fever. Both ears discharged intermittently for seven years; the left ear bothered her more. Examination shows almost normal right ear with slight retraction of vibratory membrane containing a large calcareous plaque in the posterior-inferior quadrant, while a large area of the left tympanic membrane is of a tissue-paper consistency and moves freely while the patient is talking or masticating. Under p-thiocresol treatment for two months this area became opaque, stiff, and the patient had a recession of her previous symptoms. Advised to use p-thiocresol once a month for four days.

Case 3: B. C., age 19 years, white, female, stenographer. For the last two years, following an attack of scarlet fever, the patient complained of a permanent perforation of the right tympanic membrane — she was afraid to go in swimming because of the possibility of aggravating the middle ear infection. Over a period of six weeks I carefully touched the margins of the drums with trichloracetic acid and then applied

p-thiocresol. The response was quite rapid and in two months the drum was completely healed. The scar at one point was atrophic, but continual use of p-thiocresol resulted in its thickening. Because of the smallness of the atrophic area the patient was advised to use p-thiocresol once every six months for two weeks.

Case 4: J. V., age 15 years, white, school boy. This case was brought to the clinic complaining of loss of hearing in the right ear. One year ago the patient had spontaneous rupture during an acute attack of suppurative otitis media. Examination showed a large, dry perforation of the drum. Refreshing the edges for a period of eight weeks with trichloracetic acid, followed by an external application of p-thiocresol, the opening was completely covered in four months. In this case there was also an area of atrophy.

DISCUSSION.

The successful use of p-thiocresol in four cases having deformed tympanic membranes can hardly be considered sufficient evidence to demonstrate its true value; yet, the author would like to stimulate the interest of other investigators so that this form of therapy can take its place in our armamentarium. It appeared to me that the healing of the perforations was much more rapid than what might occur under ordinary conditions. Sulphydryl compounds appear to retard the growth of granulations, especially thioglycerin, which acts also by dehydration and, therefore, should be considered as logical therapy in chronic running ears.

PARATHIOCRESOL IN POSTOPERATIVE MASTOIDITIS.

Following the sensational work with sulphydryl compounds on chronic ulcerations by Hammett and Reimann, these compounds were successfully applied to wounds and various postoperative incisions by surgeons in the Lankenau Hospital at Philadelphia. For some reason otologists did not accept this form of medication with enthusiasm and, up to now, very little work has been accomplished with the exception that p-thiocresol was used postoperatively with very poor results in simple mastoidectomies complicating scarlet fever. In all probability, the p-thiocresol was oxidized by the exudate and, therefore, lost its value as a therapeutic agent.

Dr. George M. Coates succeeded in thickening the post-auricular scar by applying p-thiocresol in lanolin base. It was this case which stimulated the author's interest in trying to find the type of cases in which this medication would be beneficial. The following is a report of 12 cases:

Case 1: R. T., age 12 years, white, school girl. Had an attack of mastoiditis during scarlet fever. Simple mastoidectomy was performed with prolonged healing over a period of two months. Parathiocresol in lanolin base applied. Patient showed marked improvement and in three weeks the post-auricular incision became closed.

Case 2: V. Z., age 10 years, white, school boy. Was subject to frequent colds and otitis media. Two months following removal of tonsils patient suffered from moderate attack of influenza, which was complicated by bilateral mastoiditis. Simple mastoidectomy done on both sides. One side was treated with p-thiocresol solution and healed 15 days before the side which received no special therapy.

Case 3: T. M., age 8 years, yellow, school boy. Unilateral mastoiditis complicating scarlet fever. Had simple mastoidectomy, but wound would not heal. After inflammatory exudate had subsided p-thiocresol was used, but the granulations appeared to diminish and treatment was discontinued. Eight months later child was seen with a small postauricular, dry fistula. Plastic was performed and p-thiocresol was used. Normal recovery took place.

Case 4: G. C., age 42 years, white, male, dentist. Suffered from mastoiditis following acute attack of tonsillitis. Simple mastoidectomy performed. Parathiocresol in lanolin base was used. Uneventful recovery in two weeks.

Case 5: W. F., age 36 years, white, male, tramp. Brought into the hospital with a septic temperature and postauricular swelling. Simple mastoidectomy with exposure of normal sinus. Uneventful recovery in 10 days with p-thiocresol solution.

Case 6: B. C., age 44 years, white, housewife. Brought to the hospital suffering from bilateral mastoiditis. Simple mastoidectomy performed. One side treated with p-thiocresol solution, which healed in less than one-half the time that it took the other side.

Case 7: P. V., age 32 years, white, housewife. Came to the clinic complaining of gradual loss of hearing and intermittent discharge from her left ear for a number of years. A simple mastoidectomy was of no avail. Examination showed a large perforation. The middle ear was dry and filled with granulations. Following radical mastoidectomy, using p-thiocresol jelly postoperatively, the patient was discharged as cured from the clinic in three weeks. Four months later there was no recurrence of her symptoms.

Case 8: S. S., age 26 years, white, male, clerk. Had suffered from pain in the right ear for five days, but the ear had been discharging intermittently for two years. There was a large perforation and the middle ear was filled with granulations. Radical mastoidectomy was finally the procedure of choice and p-thiocresol was used in the usual manner. The patient was completely cured in 13 days.

Case 9: J. R., age 11 years, white, school boy. Brought to the clinic complaining of a gradual loss of hearing. External auditory canal was filled with sessile granulations. He gave a history of having had otitis media complicating measles. Following a radical mastoidectomy with p-thiocresol as postoperative treatment the patient was completely cured in two weeks.

Case 10: J. M., age 26 years, white, male, carpenter. Came to the clinic suffering from bilateral, progressive loss of hearing and right facial paralysis of one week's standing. On further questioning he said he had intermittent discharge from both ears for five years as a result of scarlet fever. Examination showed the right ear to be filled with red, bleeding granulations. A radical mastoidectomy was performed (granulations were removed carefully from the medial wall of the middle ear cavity) and, using p-thiocresol postoperatively, the patient was discharged as healed in two weeks — there was no residual paralysis at the end of three months. Shortly after the operation on the right ear the left ear started to discharge and did not respond to conservative treatment. A simple mastoidectomy was performed and, without using the p-thiocresol, it took nearly nine weeks for the postauricular wound to heal.

Case 11: L. B., age 14 years, black, newsboy. Had all the complications of scarlet fever, including bilateral mastoiditis

with extensive destruction, necessitating operation. The patient was emaciated and, at first, was not expected to recover. Two months after discharge from the hospital he was admitted to the clinic with exceedingly stubborn, dry postauricular cavities. Using balsam of Peru over a period of four weeks stimulated granulations, following which the edges of the wound were freshened and undermined, and p-thiocresol in lanolin base was applied for two weeks until the wound had healed.

Case 12: R. M., age 43 years, white, male. Came to the clinic complaining of intermittent discharge from his right ear with progressive deafness over a period of three years. Examination showed a large perforation in the membrana tympani with the middle ear filled with granulations. Radical mastoidectomy was finally the procedure of choice. The cavity was packed with plain gauze strips infiltrated with p-thiocresol jelly. Continuing this medication daily, complete healing occurred eight days after the operation.

DISCUSSION.

The 12 cases just noted are not conclusive; yet, my results confirmed one outstanding fact; *i.e.*, sulphydryl compounds cannot be used in the presence of marked, purulent exudate; however, it is beneficial when the inflammation has subsided. This medication will not stimulate granulations for which preference should be given to balsam of Peru, etc. After the cavity has become filled with granulations, p-thiocresol can be applied, and it will be noted that the rate of epithelial growth is increased. Parathiocresol, when applied to postauricular incisions that have been sutured, will cause healing to take place in from three to four days. As regards dressings within radical mastoid cavities, epithelial growth was found more rapid than with any other form of medication that I have seen used. The author believes that, under conditions in which there is not too much exudate, the application of p-thiocresol should be the therapy of choice when epithelial proliferation is looked for.

SUMMARY.

1. The movement of the ciliated epithelium of a frog is stimulated by p-thiocresol in concentrations between 1/1,000 and 1/10,000; while at higher dilutions our experiments showed no effect.

2. Sulphydryl compounds combined with appropriate, general therapy, in all probability, will be the treatment of choice in atrophic rhinitis.

3. Parathiocresol in local application will diminish unpleasant symptoms by thickening the external surface of atrophic drums.

4. Parathiocresol increases the rate of epithelial proliferation following simple and radical mastoidectomies in the absence of marked, purulent exudate.

The author wishes to thank Dr. Stanley Reimann, Dr. T. Carrol Davis and Dr. Maurice Tainter for their help and advice; also Russel Millar, Ph.D., whose co-operation made this paper a success. Finally, the McNeil Laboratories are mentioned here for their kindness in supplying the p-thiocresol in various forms.

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NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTO-LARYNGOLOGY.

Meeting of May 18, 1938.

Affections of the Cricopharyngeal Fold. By Mr. Victor E. Negus, London, England (by invitation).

(Published in full in this issue of THE LARYNGOSCOPE.*)*

DR. CLARENCE H. SMITH, Chairman: It is my pleasant privilege to introduce a distinguished visitor from across the sea. I have tried to find some word to take the place of "distinguished" because I have heard it so often recently applied to him before different audiences; however, I can think of no other term that would so well describe him. He is a distinguished surgeon and has been for many years; he is a Master of Surgery of the University of London, a Fellow of the Royal College of Surgeons of England and attending surgeon of Kings College Hospital in London, and besides possessing the talents of his vocation, he is distinguished in another direction. In his remarkable book, "The Mechanism of the Larynx," he has established himself as an authority on the comparative anatomy and physiology of the larynx. He emphasizes in his book the action of the brain in controlling the voice as being superior to the mechanical part played by the larynx. In the introduction to this monograph by Sir Arthur Keith, he compares Mr. Negus in many ways with Darwin, and mentions that in his methods he very much resembles John Hunter.

Mr. Negus has chosen for his topic this evening, the "Affections of the Cricopharyngeal Fold." I now have the honor to present to you, Mr. Victor E. Negus, of London, England.

DISCUSSION.

DR. JOHN D. KERNAN: Mr. Negus has told you much in his short paper. At least it seemed short to me because it was so fascinating and so interesting that I did not notice the passage of time. To anyone who has done esophagoscopies for as many years as I have, the cricopharyngeal fold is the most important part of the esophagus. I have met it frequently and it has always been a headache. I look at Mr. Negus with awe because here is a man who knows all about it and who has mastered it. I never have. I always approach it with dread. Any esophagoscopy must start with the cricopharyngeal fold. It is always spastic and I always have difficulty in passing it. It is, of course, worse when it is diseased, when it is weak and easily ruptured; then the spasm is worse, and there is more danger of injury. It does not matter whether the disease is at the cricopharyngeal fold or below it. It may be a carcinoma of the stomach or carcinoma of the cardia or a gallstone; it seems as if any disease along the gastrointestinal tract will cause the cricopharyngeal fold to be tighter.

I was going to ask Mr. Negus a number of questions about it. I want to say first that so far as the comparative anatomy is concerned, that was so beautifully done, there is no use in my saying anything more about it, and the explanations he gave of the development of the protrusion are fascinating.

This fold has always seemed to me to be more prominent in women. The few cases I have seen of the Plummer-Vinson syndrome have been in anemic women who have this syndrome of anemia and difficulty in swallowing. Statistics also show that carcinoma of the mouth of the esophagus

in the region of the cricopharyngeal fold is considerably more common in women than in men. I have wondered if there is any connection between the two, if there is any biological weakness in women at this region which is not present in man.

Another thing I would like to know is this. Foreign bodies stick in the mouth of the esophagus, to be sure, but many seem to get through the cricopharyngeal fold and get stuck just below it. I have seen many foreign bodies in children, such as coins, et cetera, which seem to get through the cricopharyngeal fold and stick below it. Then our difficulty is in finding them, because the fold throws the tip of the esophagoscope over the foreign body and it is out of sight. I have seen a penny in a child which was missed twice in two examinations in spite of the fact that both times the child was under ether.

I had a question as to why diverticula occur above the fold, but Mr. Negus has answered that. Also, why do perforations occur above it? He has also answered that question. I was going to ask what is the cricopharyngeal fold? I have seen the statement made by some that it belongs to the esophagus, and by others that it is the lower part of the pharynx. If Mr. Negus says it does not matter what you call it, I am perfectly willing to take his word for it.

The lesson that we can draw from this paper is that this fold is extremely important to all of us, not only to those who are doing tube work, but to all who have to do with throat cases, in order that we may correctly interpret the early symptoms which mean later danger.

Mr. Negus, I thank you very much.

DR. CHARLES J. IMPERATORI: I will divide my remarks into two parts, Mr. Chairman, with your permission, one having to do with the paper and the other which I might just pass by as remarks.

I have always considered the cricopharyngeal fold as an exceedingly important part of the anatomy, particularly to those who are doing endoscopic work on the esophagus. What I have learned has been mostly from Mr. Negus' book and from clinical observations. I, too, have had pennies in the esophagus. I recall a case in which the Roentgenogram showed a shadow; the mother said the child swallowed a penny, the child was taken to the operating room and I did an esophagoscopy and said the penny was not there; however, to be absolutely certain, I had the child X-rayed, and the X-ray man, who was a rather excitable individual, immediately telephoned back and said the penny was still there. I re-esophagoscoped the case, and this time removed a penny. I sent the child back to the X-ray room and again the X-ray man reported that there was a penny still present. It happened that there were two pennies.

This fold of mucous membrane certainly can fool one, and as Dr. Kernan has said, particularly in doing esophagoscopy, when one passed the cricopharyngeal fold, one feels safer. In my experience, the cricopharyngeus may be called the weak spot of the esophagus and perforations of the esophagus in practically 99 per cent of the cases occur at this weak point. The esophagoscope is carried down between the esophagus and the spinal column until a point is reached where it can go no further, and the esophagoscopist wonders what is the matter. Of course, such a happening is a fatal one.

Insofar as foreign bodies becoming lodged in the region of the cricopharyngeal fold and not passing down and perforating is concerned, my experience has been that this does happen and if the esophagoscope is a small one, it is more likely to happen than if it is a larger one. If it does happen and emphysema does occur in the neck, the thing to do is to open the neck immediately for drainage and prevent a descending infection, mediastinitis and death.

The explanation that Mr. Negus made of cardiospasm seems to me to be a very ingenious one. I have given considerable thought to cardiospasm and have seen quite a number of cases, but the possibility of it being a condition, such as he explained, had never occurred to me. It certainly requires some consideration.

In demonstrating the cricopharyngeal fold, if one uses the Killian suspension, the lower hypopharynx and the cricopharyngeal fold are beautifully shown, so that you can see this crescentic fold going toward the larynx. The various hypopharyngoscopes show it, but not as well as the suspension.

I have had no experience with the Trotter operation. Dr. Orton is the one who has perfected himself in the technique of this operation, and I would rather have him do this rather extensive operation.

With your permission, Mr. Chairman, I will take this opportunity to say to Mr. Negus that we, in New York, have been highly honored to have him here to tell us about these things, and to tell him that all of us who have met him here in New York or elsewhere, have been stimulated by his papers and by him personally. I predict that in the new encyclopedias that will come out in the next few years, looking under the classification where one would look for the word "negus," one will find, first, the "negus" is a drink that one of his ancestors, Colonel Negus, devised in 1732, consisting of nutmeg, brandywine, water, sugar and lemon; second will be the "Negus" such as the Italians speak of in referring to the Emperor of Ethiopia, and somewhat confused with dictators and democracies — incidentally, the latter being a type of government that is obsolete but still slightly practiced in the United States, and third, we will find inserted "Sir Victor E. Negus, a distinguished laryngologist, a distinguished surgeon, a distinguished comparative anatomist," and the best part of all, "a fine fellow."

Dr. John M. Lore: Mr. Negus mentioned pharyngeal irritations or inflammations of the mouth or upper fold of the cricopharyngeus. I noted that he mentioned hyperkeratosis. To me that brought back memories of laryngeal conditions in which we find hyperkeratotic lesions on the cords in which we have been able to demonstrate carcinoma in many instances. I think the observations he has made and the conclusions he has drawn are very pertinent, that any inflammatory condition in this region associated with hyperkeratotic changes is malignant. We have been able to demonstrate in our laryngeal cases by diligent search of microscopic sections, tucked away in some corner of the sections, a focus showing malignant changes.

Dr. Gabriel Tucker (Philadelphia): I came over this evening to hear Mr. Negus' presentation and to see him again before he returns to England. I always learn something when I hear Mr. Negus talk and his presentation has been most delightful. I have also had the pleasure of hearing the discussion of Dr. Imperatori and Dr. Kernan, both of whom are eminent authorities on the cricopharyngeal fold. I have nothing to add by way of discussion. I came to listen and I thank you very much, Mr. Chairman, for calling upon me. It has been a very great privilege to be here with you this evening.

Dr. Robert E. Buckley: I wish also to express my appreciation and thanks to Mr. Negus for such a delightful paper.

Dr. Duncan Macpherson: I have nothing to say except to express my appreciation for Mr. Negus' paper, which was very largely over my head. I wish to thank Mr. Negus.

Mr. Victor E. Negus (London, England): I must thank you very much, Mr. President, ladies and gentlemen, for being so receptive to my paper. I can assure you it has been a great pleasure to me to give it. There are one or two points I want to mention.

As to the question of injury, I did not speak of traumatic perforation of the mouth of the esophagus. I know that it does happen. The way I try

to avoid it myself is to have a definite routine for passing the esophagoscope. I pick up the right cartilage of Wrisberg within the aryepiglottic fold, pass the esophagoscope down to the right or lateral side of the aryepiglottic fold and then bring it slightly to the midline, when it should pass quite easily into the esophagus. If that is neglected, the beginner may find he is in the wrong pyriform fossa and by pushing to the left instead of to the right, he will cause a perforation. If the esophagoscope is in the right position, the cricopharyngeal fold will relax before you without any danger. We had a laryngologist visiting in London once from Bulgaria who had a method of passing the esophagoscope very rapidly. He said he thought it was a mistake to think too much of the anatomy of the region, but for most of us it would be well to remember the anatomical details.

I do not know of any reason why hypopharyngitis should occur more frequently in women. There is no anatomical reason that I know of. Of course, there is also the difficulty of explaining why cancer of the larynx should occur more frequently in men.

Concerning the question of a foreign body below the fold, my experience has been that a large foreign body, such as a penny, may pass through the cricopharyngeal fold of a small child and then become impacted at the upper thoracic inlet where the clavicles come in and articulate with the sternum, and in a small child this inlet is more difficult to pass than the cricopharyngeal fold itself. I have heard another story of the two pennies. The X-ray man is said to have reported a penny in the esophagus. A tube was passed and a halfpenny removed. Later another halfpenny was removed. He did not bother to report two halfpennies, but just a penny.

Reading a paper after dinner is sometimes difficult. I hope that I did not leave out too much. I certainly thank Dr. Imperatori very much indeed for his kind remarks.

Dr. Tucker said he had nothing to say. I am tonight merely repeating what I learned from him. When I came to Philadelphia 15 years ago, my knowledge of the esophagus was practically nil. He was my teacher in association with Dr. Chevalier Jackson. I am merely repeating the teachings I derived from him and I am extremely grateful to him. I hope he realizes that.

Hyperkeratosis, I believe, has an influence on the development of cancer. I have removed the mouth of the esophagus in a patient with signs of keratosis and found malignancy present. In a patient with inflammation and ulceration, who is unable to swallow, there may be no alternative to operating before there are actual demonstrable malignant changes deep in the tissues. It may be better, in certain cases, to remove the mouth of the esophagus before cancer is actually present. If it is left until the disease is far advanced, the results of operation are poor.

I wish once more to thank you all very much indeed.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

SECTION ON OTOLARYNGOLOGY.

Meeting of Oct. 21, 1938.

Hemangio-Endothelioma. Case Report. By Dr. John J. Hochfilzer. Lantern slides were shown.

The case presented this evening, a woman, age 58 years, was seen at the Out-Patient Department of the Ancker Hospital four weeks ago. She gave the following history:

About 1928, patient had an attack of "gallbladder trouble," probably colic. Soon afterwards, she attempted to use a telephone, only to find that she was unable to hear out of her left ear. She states she was able to hear well before. Her condition of deafness persisted without changes. About 1934 or 1935, serous discharge, which was never bloody or purulent, began. This discharge was odorless and it was never profuse.

In 1936, patient noticed a "lump" projecting from her left external auditory canal. The mass was of moderate consistency and was not tender. It did not bleed even on palpation. She believes the mass has enlarged gradually. In about September, 1937, patient, while looking into a mirror, noticed that her left upper eyelid seemed to droop, and that the left angle of her mouth seemed drawn down. After that she found that the left side of her face felt weak, but not numb. Her tongue began to feel numb and weak; it seemed "awkward" in that it did not move "quite the way she wanted it to." There was no previous "cold," infection, etc. Her facial weakness has not changed, but her tongue has improved. There was no swalling difficulty or anesthesia or paresthesia of the throat. In August, 1938, patient noticed hoarseness of her voice. She did not have a "cold" or laryngitis previously. Her hoarseness has persisted unchanged.

Examination revealed the following findings: Right ear, negative; left ear, the external auditory canal was completely filled out by a protruding tumor which looked like a very large ear polyp. The surface was epithelized, the consistency rather hard; with a fine probe one was able to separate the tumor from the canal wall quite a way in, but one was unable to determine from where the tumor originated. Patient hears a whispering voice at 4 feet in that ear. Nose and pharynx were negative. Indirect laryngoscopy showed a complete fixation of the left vocal cord. A slight paresis of the left facial nerve in all its branches was also present. We were under the impression that we were dealing with an unusually large ear polyp, which had developed in the course of a long, persisting, chronic, suppurative otitis media. An attempt of a partial removal for biopsy purposes was made at the Dispensary, but was unsuccessful because of intolerable pain. Dr. Delavan removed the tumor several days later and will give you further details about it.

Dr. Nobel, Pathologist of the Hospital, gave the following report of the microscopic sections: "Sections of the tissue from external auditory canal show the surface of the lesion to be slightly ulcerative. The tumor itself is composed of numerous blood spaces lined by endothelium. In places, this endothelium is fairly flattened, but for the most part it shows definite evidence of proliferation. Mitotic figures are not numerous, but are definitely present. The histologic malignancy of the lesion is not great, but due to its position, its complete surgical removal will in all probability be difficult. The character of the lesion suggests the possibility of radiation sensitivity.

Diagnosis: Hemangio-Endothelioma.

X-ray report, Oct. 17, 1938: Right mastoid shows normal pneumatization and density, with a small type of cell predominating. Left mastoid shows a moderate degree of pneumatization centrally, with small cells predominating, suggesting mastoiditis during development period, with generalized sclerosis and incomplete pneumatization resulting. There is considerable enlargement of the calibre of the external auditory canal, with extension medially of this enlargement, showing rather sharply defined borders. This destruction evidently irradiates the anterior plate of the lateral sinus. The lateral sinus itself is rather large in calibre and extends rather considerably anteriorly. The degree of extension medially in the petrous portion and internal canal is not well demonstrated for the process involving the external canal, but there is some evidence that this lesion does extend into this area. These findings could be due to cholesteatoma or other tumor of an expanding type. Films of skull show no evidence of other cranial or intracranial pathology.

A hemangio-endothelioma is an angiomatous tumor possessing low grade malignancy, presenting a characteristic clinical appearance and having a precise histologic structure. It is composed essentially of blood spaces and vessels in different stages of development. The blood does not circulate through the tumor mass because of the immaturity of many of the vessels.

I have found only one case reported in the literature; therefore, hemangio-endothelioma of the temporal bone must be a rather late occurrence, and so very little is known in regard to the prognosis and treatment. Apparently this case shows only a very low grade of malignancy, judging from the slow progress and rather small amount of destruction done so far. The paralysis of the VIIth and Xth nerves may be explained by the evidence of bone destruction as shown in the X-ray film, involving region of the posterior wall of the auditory canal and probably also the sinus plate, this being the region in which the facial and vagus nerve take their course.

But as the pathologist indicated in his report, these tumors are radiosensitive; therefore, X-ray therapy or implantation of radon seeds will probably be the best therapy.

DISCUSSION.

DR. PHILIP A. DELAVAN: It was my misfortune to have removed this tumor. The canal was packed so tightly with the tumor that a snare wire could not be gotten down to the pedicle. A biting forceps was used and gradually worked over the mass until we thought we were in contact or should be in contact with the drum. The forceps was then closed and the tumor removed. It was really quite terrifying to see the amount of blood that gushed out of the external auditory canal. The carotid artery was compressed, but this did not make any appreciable difference in the amount of blood. With steady pressure and adrenalin packs, the bleeding was finally controlled. A tight pack was then put in and left for three or four days. This was then cautiously removed and the bleeding recurred.

It was again packed and left for three or four days more, and gently removed. No bleeding occurred at this time. The pack was left out and there has been no bleeding from the ear for about two weeks. As you look into the ear at the present time, the tumor mass is again enlarging and packed in a purulent discharge.

Upon examination of the nasopharynx with a nasopharyngoscope, a small tumor mass can be seen protruding through the Eustachian orifice. There has never been any history of bleeding from the throat.

The Nasal Accessory Sinuses as Foci of Infection in Arthritis. By Dr. H. L. Williams.

Summary: Although in an unselected group of 100 cases of infectious arthritis, 20 were found to be cases of clinically proven sinus disease; the results experienced by the 12 patients who were operated on do not support the conclu-

sion that sinusitis is the principal focus of infection in cases of infectious arthritis. If disease of the sinuses is found, an attempt should be made to eradicate it, but too optimistic an outlook as to the results of this type of therapy should be avoided. It would seem to be definitely an error, however, to assume that the sinuses can be ignored as possible foci of infection in cases of infectious arthritis.

DISCUSSION.

DR. CHARLES H. SLOCUMB (by invitation): There is much disagreement regarding the beneficial effects of the treatment of foci of infection in cases of infectious arthritis and yet, from the practical therapeutic standpoint, most physicians attempt to eradicate foci of infection in such cases, not because of the many patients who notice no improvement or only slight improvement in their joints after such treatment, but because of the few who notice much improvement or because occasionally the patient is cured by such a procedure, especially if treatment is given early in the course of the disease. By removing foci in which infection can be demonstrated in cases of infectious arthritis, the opportunity of possibly stopping the progress of the arthritis is created. If the patient is not fortunate enough to be helped by such treatment, he is not in a worse condition after such treatment. Reactions after removal of such foci are rarely seen in cases of infectious arthritis if the general condition warrants such treatment or operative procedure, and if the usual precautions are observed, such as removing a focus of infection at the proper time — for instance, not removing tonsils in the presence of acute inflammation.

Approximately a third of the patients who come to a physician complaining of arthritis or rheumatism have infectious arthritis. Infectious arthritis tends to progress from joint to joint, causing permanent damage of the involved joints. Although the removal of foci of infection alone is not adequate without employing other measures to protect against deformities of joints and to build up general resistance, treatment of foci of infection has a definite place in the regimen of treatment for such patients. Dr. Williams has shown in this series that significant infection in sinuses should be dealt with as other foci of infection are treated, although significant sinus infection is not a common focus among patients who have infectious arthritis.

BOOK REVIEWS.

Nasal Tuberculosis and Tuberculids. By J. Margarot and J. Terracol, Professors of the Medical Faculty of Montpellier. 356 pages, including the bibliography and index, with 38 illustrations. Bordeux: Impriemeries Delmas, 6, place Saint-Christoly, 1938. Price, 60 Francs.

The general problem of tuberculosis is first presented, followed by the history of the disease. The morphological and biological variations of human and bovine tubercle bacilli receive due consideration. Typical nasal tuberculosis, the general etiology and pathology, general anatomic forms, such as ulceration, tubercle with caseation, lupus of the skin of the nose in its various forms, such as erythematous, nonulcerated and ulcerated, are discussed in detail. Tuberculosis of the pituitary membrane, of the mucous membrane, tuberculomata, ulcers, tuberculosis of the bones of the nose and sinuses are presented.

Under the name of Besnier-Boeck disease are grouped chronic infectious granulomata presumably caused by an attenuated tuberculous virus affecting the skin, the internal organs, the lymphatic system and the skeleton.

The infection of the lymph nodes, lymph and blood stream metastasis, antituberculous prophylaxis, diagnosis of the different types of lesions, the prognosis and treatment all receive adequate attention. The treatment includes chemotherapy, tuberculin, vaccines, allergins, antigens, sera, drugs, such as iodine, cod liver oil and arsenic, diet, climatic treatment, heliotherapy, antisyphilitic therapy for patients with tuberculosis and syphilis, treatment of secondary infections, etc.

An abstract of the monograph is printed in German, for those who read German and do not read French, and an abstract in English; however, both the German and the English abstracts are most too brief to present adequately the detailed information as printed in French.

The monograph lives up to the thoroughness with which our best European confreres present their subjects. It is very complete in every detail, and leaves very little to be desired in such a treatise. It is a credit to our French confreres.

An American reviewer cannot help being impressed by the fact that there are a few less than 100 references to the French literature, a few less than 20 to the German, with only five Italian, three Spanish, two Swiss, two British, two Scandinavian, one from the Netherlands and not any from either Russia or America.

The illustrations are in black and white, but these are well done. The legends are brief, but very descriptive. Could these have been done in color the illustrations would be perfect. F. R. S.

Fractures of the Jaw. By Robert H. Levy, M.D., M.D.S., F.A.C.S., Professor of Maxillo-Facial Surgery, School of Medicine and Graduate School of Medicine, and of Clinical Maxillo-Facial Surgery, School of Dentistry, University of Pennsylvania; Chief of Maxillo-Facial Surgery, Graduate Hospital; Consultant in Plastic Surgery, Children's Hospital; Oral Surgeon to the Presbyterian Hospital; Colonel, Medical Officers Reserve Corps, U. S. Army; and Lawrence Curtis, A.B., M.D., D.D.S., F.A.C.S., Assistant Professor of Maxillo-Facial Surgery, Graduate School of Medicine and School of Dentistry, University of Pennsylvania; Visiting Chief of Maxillo-Facial Surgery, Philadelphia General and Delaware County Hospitals; Associate in Oral Surgery, Presbyterian Hospital. Second Edition, thoroughly revised, with 192 pages, including index and 199 engravings. Philadelphia: Lea & Febiger, Washington Square, 1938. Price $4.50.

This excellent monograph by two eminent authorities re-emphasizes a simplified technique in the treatment of fractures of the jaw. For the general surgeon or specialist this offers clear, concise and detailed descriptions and excellent illustrations, making possible a more intelligent handling of the uncomplicated case.

This book should be in the library or available to every postgraduate student or otolaryngologist, since in this day of aeroplane and automobile he will be confronted with an increasing number of cases of trauma about the head and neck. We recommend it as a real contribution to the otolaryngologist's armamentarium. A. M. A.

Diseases of the Ear, Nose and Throat. By Francis L. Lederer, B.Sc., M.D., F.A.C.S., Professor and Head of the Department of Laryngology, Rhinology and Otology, University of Illinois College of Medicine, Chicago; Chief of the Otolaryngological Service, Research and Educational Hospital. Illustrated with nearly 500 halftones and engravings, mostly original, and 16 full-page color plates. Contains 885 pages with index. Philadelphia: F. A. Davis Company, 1914-16 Cherry street, 1938. Price $10.00.

Many in the Ear, Nose and Throat field have felt the need of a book so complete that one need not consult other texts for fundamental data. We are particularly impressed by the legends explaining the practical importance of certain anatomical structures; such correlation is bound to make more interesting reading, especially in histology and anatomy.

The drawings, which are colored in most instances, show the fine hand of the "Tom Jones" school of medical craftsmanship; the unique method of supplementing the colored gross drawings with histological inserts will prove a crutch to many a limping memory.

The fine colored photographs of laryngeal pathology by Alfonso Shandl, of the Hajek Clinic, certainly present clinical laryngology with definite certainty.

The book is replete with a multitude of differential diagnosis tables which show great ingenuity. In fact, the entire book seems to have been written with a view of covering the specialist's field and its overlappings as exemplified by a splendid chapter on Correlated Conditions, followed by one on Facial Neuralgias, and still another on Enlargements of the Neck. A short chapter on psychiatric aspects, coupled with an unusually complete resume of blood dyscrasias enlarges one's outlook on the book's value.

We cannot help but notice, from the wealth of photographs of unusual bizarre types of cases, that the author certainly has seen the seamy side of oto-rhino-laryngology, demonstrating the fact that the specialty has made many strides since the days of galvano-cautery and the guillotine.

To summarize, this textbook embodies originality, clarity of subject-matter, unusually well adapted illustrations of special pathologies as presented in a series of selected photographs and a terse style, all of which commend it not only to the busy otolaryngologist, but also to the interested general medical profession. E. L. M.

THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

The Section Meetings of the Society are scheduled as follows:

Eastern Section: Boston, Jan. 11, 1939.

Southern Section: New Orleans, Jan. 14, 1939.

Middle Section: Sioux City, Iowa, Jan. 19-20, 1939.

Western Section: Spokane, Jan. 29, 1939.

Subject to the confirmation of the Council, the annual meeting will be held at the Drake Hotel, Chicago, on May 9, 10 and 11, 1939. Dr. Lillie wishes to repeat that the program for this meeting is to be eminently practical and informal, and it is especially desired that there be free discussion from the floor.

For those desiring to present papers at either the Section or the annual meetings, we should like to state that all programs are formulated far in advance. The 1939 annual program, for example, was completed in August, 1938.

DR. C. STEWART NASH, *Secretary.*

OFFICIAL ORGAN
AMERICAN OTOLOGICAL SOCIETY, Inc.

Vol. XLVIII DECEMBER, 1938. No. 12

THE LARYNGOSCOPE

AN INTERNATIONAL MONTHLY JOURNAL
DEVOTED TO DISEASES OF THE

EAR - NOSE - THROAT

FOUNDED IN 1896 BY

DR. MAX A. GOLDSTEIN
Managing Editor and Publisher

For Contents See Page 1.

Subscription, $6.00 per Annum, in Advance.

Foreign Subscription, 25 Shillings per Annum, Post Free.
Single Copies, 75 cents.

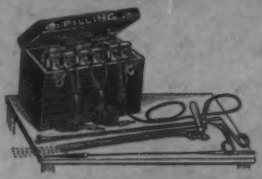
PUBLISHED BY THE LARYNGOSCOPE CO.

4574 West Papin Street St. Louis, Mo., U.S.A.

FOREIGN OFFICE, BAILLIERE, TINDALL & COX
7 & 8 HENRIETTA ST., COVENT GARDEN, LONDON, ENG.

(Entered at the Postoffice at St. Louis, Mo., as Second Class Matter, in July, 1896)

INDEX NUMBER



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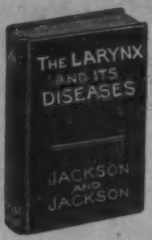

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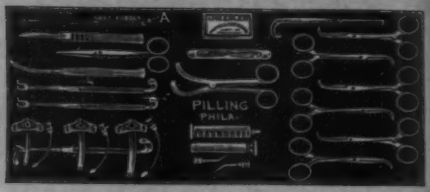

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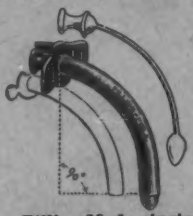

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